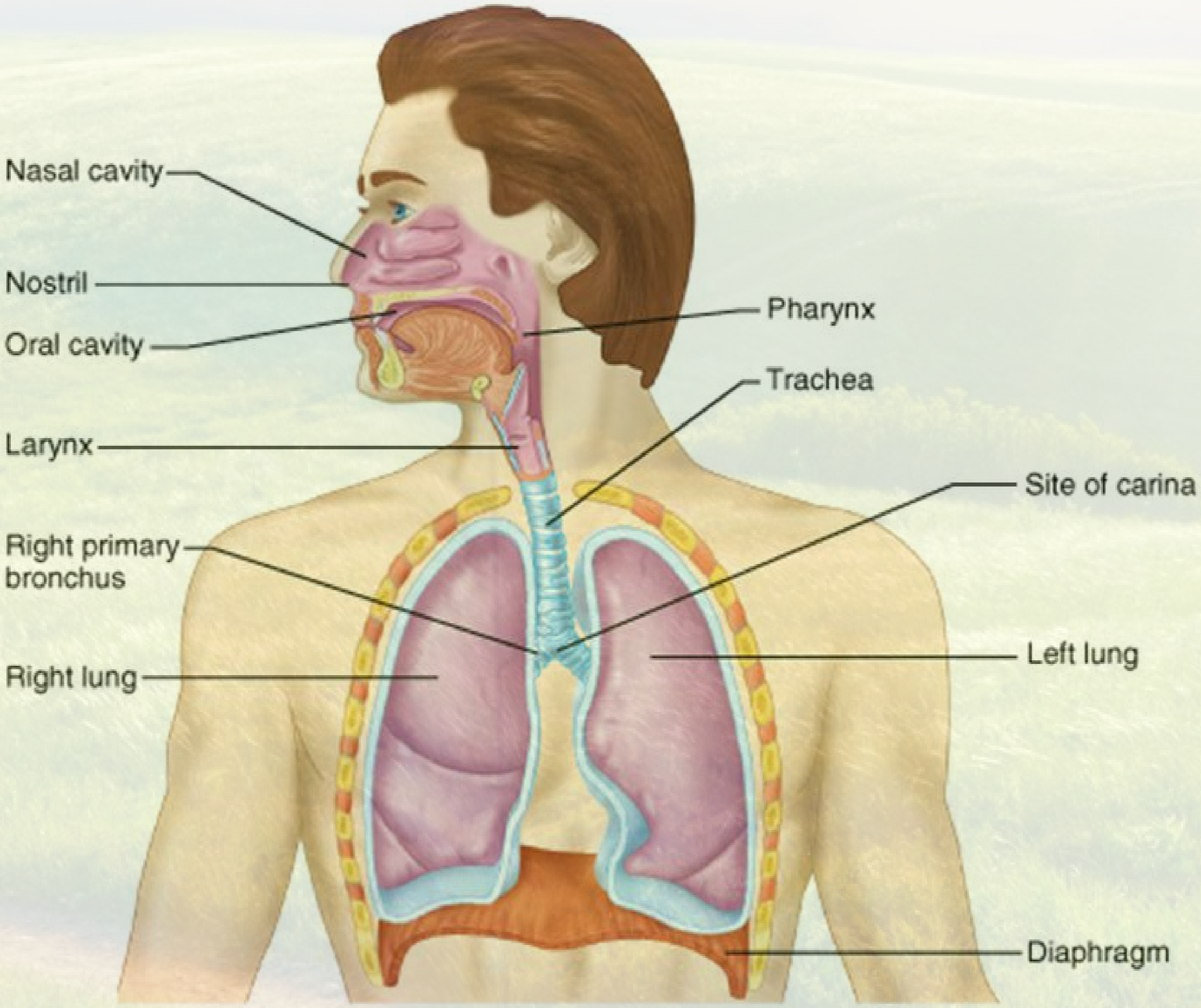
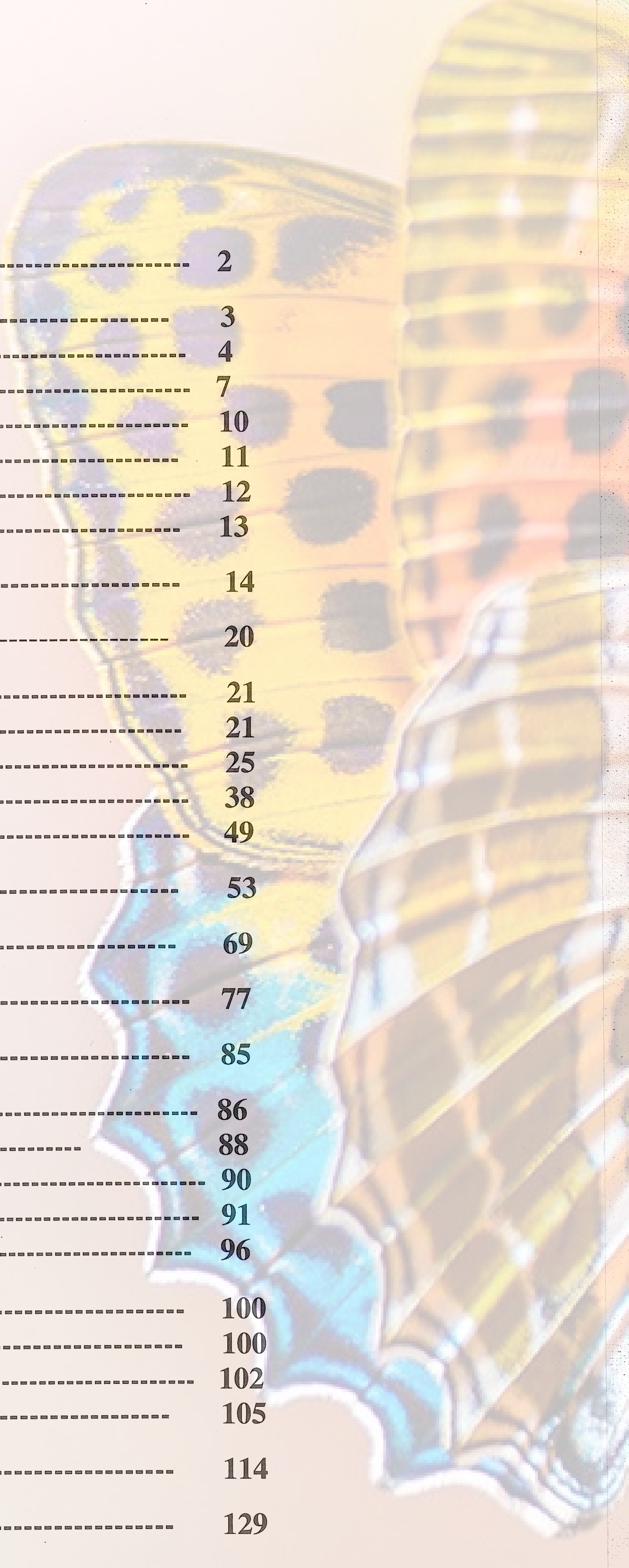
Respiratory System


Nasal cavity
Nostril
Oral cavity
Larynx
Right primary bronchus
Right lung
Pharynx
Trachea
Site of carina
Left lung
Diaphragm



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INTRODUCTION

CLASSIFICATION OF CHEST DISEASES

1. BRONCHIAL DISEASES:

- Bronchitis (*acute* & *chronic*).
- Bronchial asthma.
- Bronchiectasis.
- Bronchogenic carcinoma.

2. PARENCHYMATOUS LUNG DISEASES:

- Consolidation (*pneumonia*).
- Cavitation (*lung abscess*).
- Collapse.
- Fibrosis.
- Emphysema.
- TB.

3. PLEURAL DISEASES:

- Pleurisy.
- Pleural effusion.
- Pneumothorax.
- Hydropneumothorax.

4. MEDIASTINAL DISEASES.

5. DIAPHRAGMATIC DISEASES.

6. CHEST WALL DISEASES.

CHEST SYMPTOMS

- COUGH
- HEMOPTYSIS
- CHEST PAIN
- DYSPNEA

COUGH

DEFINITION

- ***Cough*** is an explosive expiration that provides a normal protective mechanism for clearing the tracheo-bronchial tree of: secretions & foreign material.
- It is the most common symptom of chest diseases.

ETIOLOGY

I. Reflex cough: (due to irritation of cough receptors)

A. Respiratory causes:

1. *Airway diseases:*
   - **I**nflammation: Pharyngitis, Laryngitis, **BRONCHITIS**, **BRONCHIECTASIS**.
   - **I**nfiltration: Sarcoidosis, **BR. CARCINOMA**.
   - **C**onstriction: **COPD**, **BR. ASTHMA**.
   - **C**ompression: Mediastinal masses, LN, AA (**Brassy cough**).
2. *Parenchymatous lung diseases:*
   - Pneumonia, Lung abscess, Fibrosis, TB.
3. *Pleural diseases:*
   - Pleurisy, Pleural effusion, Pneumothorax, Hydropneumothorax.

B. Extra-respiratory causes:

1. **Cardiovascular diseases:**
   - Pulmonary congestion: *Left – sided heart failure*.
   - Pulmonary embolism.
2. **Other causes:**
   - Abdominal causes: Subphrenic abscess, GORD.
   - DRUGS: ACE – I.

II. Central cough: (due to stimulation of the cough center)

- Brain tumours, Encephalitis, CVS.

III. Psychogenic (Hysterical) cough:

- Usually: dry cough in young neurotic females.

APPROACH TO THE PATIENT

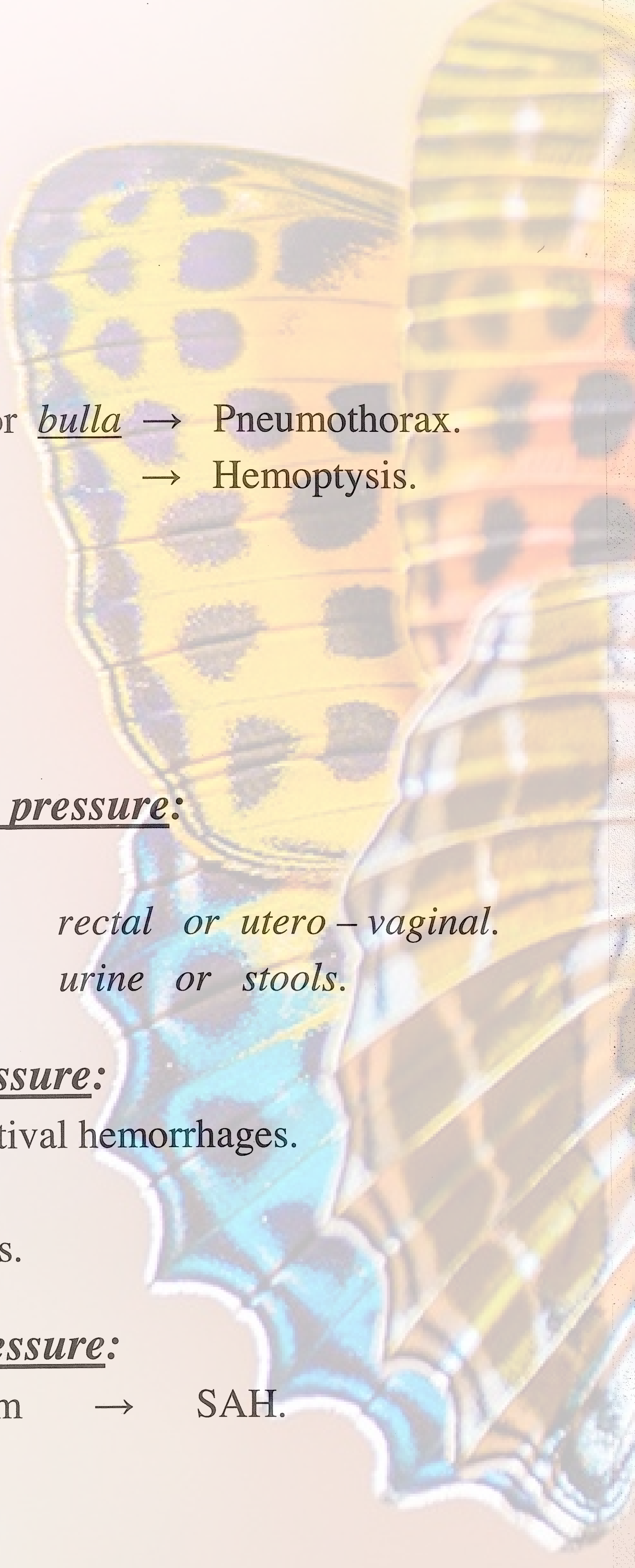
1. **Is the cough ACUTE or CHRONIC ??**
   - ACUTE (< 3 weeks): **e.g.** Pneumonia, Pulmonary embolism.
   - CHRONIC (> 3 weeks): **e.g.** COPD, GORD.

2. **Is the cough DRY or PRODUCTIVE ??**
   - DRY: **e.g.** Pleurisy, Psychogenic, DRUG (ACE – I).
   - PRODUCTIVE: **e.g.** Chronic bronchitis, Bronchiectasis, Lung abscess.

3. **Is there PERIODICITY ?? (DIURNAL or SEASONAL)**
   - DIURNAL
     - (Early morning): **e.g.** **Br**onchitis, **Br**. asthma, **Br**onchiectasis.
     - (Nocturnal): **e.g.** **Cardiac** asthma.
   - SEASONAL (winter): **e.g.** **Br**onchitis, **Br**. asthma, **Br**onchiectasis.

4. **Is there associated WHEEZING ??**
   - GENERALISED: **e.g.** **Br**onchitis, **Br**. asthma, **Br**onchiectasis, COPD.
   - LOCALISED: **e.g.** **Br**. carcinoma.

5. **Is the patient taking an ACE – I ??**



COMPLICATIONS [*]

I. Thoracic

1. Rupture of a pulmonary *bleb* or *bulla* → Pneumothorax.
2. Rupture of bronchial varices → Hemoptysis.
3. Rupture of an aneurysm.
4. Stress fracture of a rib.

II. Extrathoracic

1. *Increased intra-abdominal pressure*:

- Hernia.
- Prolapse: *rectal or utero – vaginal.*
- Stress incontinence of: *urine or stools.*

2. *Increased intra-ocular pressure*:

- Retinal & subconjunctival hemorrhages.
- Retinal detachement.
- Puffiness of the eye lids.

3. *Increased intra-cranial pressure:*

- Rupture of an aneurysm → SAH.
- Cough syncope.

4. *OTHERS*:

- Insomnia.
- Cough – induced vomiting.

---

[*] You have a cough? Go home tonight, take a whole bottle of Picolax; a potent laxative, tomorrow you'll be afraid to cough… ~ **Pearl Williams**

HEMOPTYSIS

DEFINITION

- Coughing of blood originating from: ***below the vocal cords***.
- Bleeding originating from above the vocal cords is known as: false hemoptysis.
- Hemoptysis may be: *frank or blood tinged*.

ETIOLOGY

(1) **Respiratory causes**:

A. *Laryngeal causes*:
- Inflammation.
- Tumours.

B. *Tracheo-bronchial causes*:
- Acute **B**ronchitis. (**the most common cause**)
- **B**ronchiectasis.
- **B**ronchial carcinoma.
- **B**ronchial adenoma.

C. *Pulmonary causes*:
- Pulmonary TB.
- Pulmonary embolism.
- Pneumonia.
- Lung abscess.
- Goodpasture syndrome.

(2) **Cardiovascular causes**:

- Pulmonary congestion, **e.g.** MS & acute pulmonary oedema.
- Pulmonary infection.
- Pulmonary infraction.
- Rupture of bronchial varices.
- Rupture of aortic aneurysm.

(3) **Systemic diseases**:

Hemorrhagic blood diseases, **e.g.**
- Hemophilia.
- Purpura.

MANAGEMENT OF HEMOPTYSIS

I. Diagnosis:

1 Differentiation between hemoptysis & hematemesis:

	Hemoptysis	Hematemesis
Past history	Chest disease	GIT disease
The attack	Cough	Vomiting
Blood	Bright red, frothy	Dark red, food particles
After the attack	Streaked sputum	Melena
Examination	Chest signs	Abdominal signs
Investigations	Chest or heart disease	GIT disease

2 Exclusion of false hemoptysis:

- Examination of the upper resoiratory tract usually reveals the cause.

3 Detection of the cause of hemoptysis:

A) Clinical picture:

- Symptoms & Signs.

B) Investigations:

Chest imaging:	CXR, CT.
Cardiac investigations:	ECG, Echocardiography.
Laboratory investigations:	CBC, Bleeding profile, sputum analysis.
BRONCHOSCOPY.	

II. Treatment:

1. Treatment of the cause.

2. Cases of massive hemoptysis require:
    - *Hospitalization.*
    - *Sedation.*
    - *Blood transfusion & anti-shock measures.*

CHEST PAIN

ETIOLOGY

I. Cardiovascular causes:

- **Myocardium:** CAD *(angina or infarction).*
- **Pericardium:** Pericarditis or pericardial effusion.
- **Endocardium:** Mitral valve prolapse.
- **Aorta** Aortic aneurysm & Aortic dissection.
- **Pulmonary:** Pulmonary embolism & Pulmonary infarction.
- **Pain of cardiac neurosis.**
- **Huge cardiomegaly.**

II. Respiratory causes:

- Pleural disease: *pleurisy, pleural effusion, pneumothorax, hydropneumothorax.*
- Pulmonary disease extending to the pleura , e.g. *pneumonia & lung abscess.*
- Acute massive lung collapse.

III. Chest wall causes:

- Skin: wounds.
- Breast: mastitis, tumour.
- Ribs: osteomyelitis, tumour, fracture, Tietze syndrome.
- Intercostal muscles: myositis, muscle strain (from severe cough).
- Intercostal nerves: nerve root pain (herpes zoster).

IV. Mediastinal causes:

- Tumours, LN, Mediastinitis.

V. GIT:

- GORD, peptic ulcer, cholecystitis.

CAUSES OF ACUTE CHEST PAIN

(1) MYOCARDIUM: *angina pectoris* & *acute myocardial infarction*.

(2) PERICARDIUM: *acute pericarditis*.

(3) ENDOCARDIUM: *mitral valve prolapse*.

(4) AORTA: *dissecting aneurysm of the aorta*.

(5) PULMONARY: *massive pulmonary embolism* & *pulmonary infarction*.

(6) CARDIAC NEUROSIS.

(7) Pleural disease: *acute pleurisy* & *pneumothorax*.

(8) Lung disease: *acute massive lung collapse*.

(9) Musculo-skeletal disorders: *Tietze syndrome*.

(10) Nerve root pain: *Herpetic neuralgia*.

(11) GIT: *GORD*, *peptic ulcer*, *cholecystitis*.

DYSPNEA

ETIOLOGY

1. Cardiovascular diseases

1. **P**ulmonary congestion: *Left – sided heart failure*.
2. **P**ulmonary embolism.
3. **P**ericardial effusion.

2. Chest diseases

1. Laryngeal:

- Foreign body, Tumours.

2. Tracheo-bronchial:

- **Br**onchitis, **Br**. asthma, **Br**onchiectasis, COPD.

3. Lung:

- Consolidation, Collapse.
- Fibrosis (pulmonary, ILD).

4. Pleural:

- Pleurisy, pleural effusion, pneumothorax, hydropneumothorax.

5. Chest wall:

- Chest deformities, scleroderma, marked obesity (*Pick syndrome*).

3. Abdominal diseases

- Abdominal distension, **e.g.** marked ascites.

4. General diseases

- Anemia.
- Hemorrhage & shock.
- Acidosis.

5. Neurological diseases

1. *Diaphragmatic paralysis*.
2. *Disorders of neuromuscular apparatus*:
    - CNS: Head injury, CVS, Drugs (*opiates & barbiturates*).
    - AHCs: MND, Poliomyelitis.
    - Peripheral nerve: Polyneuropathy, **e.g.** *GBS*.
    - NMJ: Myasthenia gravis.
    - Muscle: Myopathies.

6. Psychogenic (Hysterical) dyspnea.

CAUSES OF ACUTE DYSPNEA

1. **Acute** massive myocardial infarction, (Acute LVF).
2. **Acute** pulmonary embolism.
3. **Acute** massive lung collapse.
4. **P**neumothorax (Tension).

4 X 4

5. **P**neumonia.
6. **P**leurisy.

7. *Bronchus:* Bronchial asthma.
8. *Larynx:* Inhaled FB, *laryngeal spasm*, *laryngeal oedema*.

9. **H**emorrhage, shock, acidosis.
10. **H**ysterical.

CHEST EXAMINATION

INSPECTION

1. Shape:

1. *Symmetrical:*
   - Barrel chest: emphysema.
2. *Asymmetrical:*
   - Unilateral bulge: pleural effusion, pneumothorax.
   - Unilateral retraction: lung collapse, lung fibrosis.

2. Respiratory movements:

- In different chest diseases, respiratory movements are limited.

PALPATION

1. Trachea:

- To assess whether the trachea is **central** or **shifted** to one side:
  - *Shift to the same side of the disease*:
    Occurs in the same causes of: unilateral retraction.
  - *Shift to the opposite side of the disease*:
    Occurs in the same causes of: unilateral bulge.

2. Tactile Vocal Fremitus (TVF):

- *Causes of increased TVF:*
  - Consolidation, e.g. pneumonia.
  - Cavitation, e.g. lung abscess.
  - Cavitation surrounded by consolidation, e.g. bronchiectasis.
- *Causes of decreased TVF:*
  - Almost all other chest diseases.

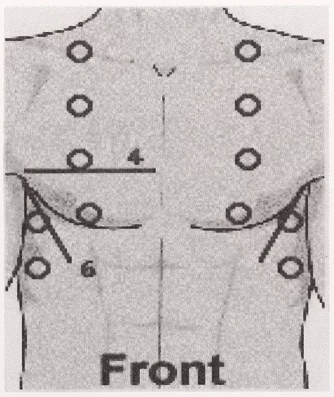
PERCUSSION

Percussion notes in the chest	
Note	**Disease**
Resonance	Normal
Hyper-resonance	Hyperinflation as in asthma & emphysema Pneumothorax Very thin people
Tympanitic resonance	Tension pneumothorax
Dullness	Consolidation Cavitation Collapse Fibrosis
Stony dullness	Pleural effusion

Causes of Dullness over the Base of the Right lung:

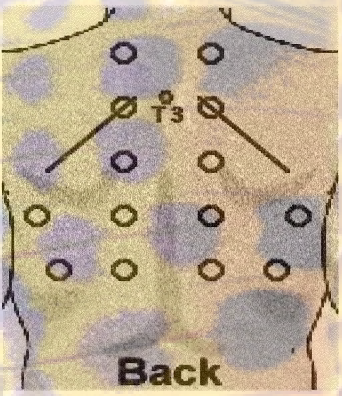
1. **Supra-diaphragmatic causes**:
   - Pleural disease: *pleural effusion, hydropneumothorax.*
   - Basal lung disease: *consolidation, caviation, collapse, fibrosis, bronchiectasis.*
   - Heart disease: *huge pericardial effusion.*
2. **Diaphragmatic paralysis**.
3. **Infra-diaphragmatic causes**:
   - *Acute liver abscess.*
   - *Subphrenic abscess.*
   - *Marked ascites.*
   - *Pregnancy.*
   - *Huge abdominal mass.*
4. **Chest wall diseases**:

   e.g. tumours.



Points for:

Percussion
&
Auscultation



AUSCULTATION

I. BREATH SOUNDS

A) Types

1. Normal Vesicular Breathing:

- It is heard normally in: NORMAL persons.

2. Vesicular Breathing with prolonged expiration:

- It is characterized by: prolonged expiration.
- It is heard in: BRONCHIAL NARROWING.

3. Bronchial Breathing:

- It is characterized by: hollow sound (due to good conduction).
- It is heard in: CONSOLIDATION, CAVITATION.
- It is confirmed by: ***bronchophony, aegophony, whispering pectorlioquy.***

B) Intensity

1. Decreased:

- Physiological: overweight persons.
- Pathological: collapse, fibrosis, pl. eff, pneumothorax, emphysema.

2. Increased:

- Physiological: very thin chest wall.
- Pathological: causes of bronchial breathing.

II. ADDITIONAL SOUNDS

A) Crepitations *(Crackles or Rales)*

Definition

- These are interrupted (non-continuous) sounds produced by:
  - Passage of air through fluid in the respiratory passages, OR:
  - Pulmonary fibrosis.

Types

a) Fine:
- Character: **like the sound of rolling a tuft of your hair between your fingers near your ear.**
- Heard in: LVF (basal & bilateral), **TB** (apical), **Early Pneumonia.**

b) Medium-sized: " Consonating or Non-consonating"
- Character: **like the sound of a crack.**
- Heard in: **Bronchiectasis, Lung abscess, Pneumonia, Fibrosis.**

c) Coarse:
- Character: **like the sound of loud gurgling & bubbling.**
- Heard in: **Bronchiectasis, Lung abscess, Pulmonary oedema.**

Timing

- They are usually: INSPIRATORY.

Site

- Localized: e.g. simple secretion, FB, tumour.
- Generalized: e.g. pulmonary oedema.
- Bilateral & Basal: e.g. LVF (fine), bronchiectasis (medium or coarse).
- Kronig's isthmus: e.g. TB fibrosis.

Persistence after cough

- *If you hear crepitations, ask the patient to cough & then auscultate again:*

- If sounds persist = organic pathological lesion.
- If sounds dissapear: = simple secretion.

B) Wheezes *(Rhonchi)*

Definition

- These are continuous sounds produced by:
  - o Passage of air through the narrow bronchi *e.g. COPD, Br. asthma*.

Types

a) Sibilant: high pitched due to narrowing of the SMALL bronchi.

b) Sonorous: low pitched due to narrowing of the BIG bronchi.

Timing

- They are usually: EXPIRATORY.
- In severe obstruction: they may be heard also INSPIRATORY.

Site

- o Localized: e.g. simple secretion, FB, tumour.
- o Generalized: e.g. COPD, bronchial asthma.
- o Kronig's isthmus: e.g. Pancoast tumour.

Persistence after cough

- *If you hear wheezes, ask the patient to cough & then auscultate again*:

- o If sounds persist = organic pathological lesion.
- o If sounds dissapear: = simple secretion.

C) Pleural rub (see later).

Despign sign

- Normally: on the back you hear BB till the 2nd dorsal spine in adults.
- + ve sign: on the back you hear BB below the 2nd dorsal spine in adults in the presence of enlarged mediastinal lymph nodes (they act as good conducting medium).
- Confirm by: vocal resonance (bronchophony) & whispering pectorliquy.
- Value: it indicates the presence of ***enlarged mediastinal lymph nodes***.

Local signs of chest disease

Chest disease	Inspection		Palpation		Percussion	Auscultation	
	Shape	Movement	Trachea	TVF	Note	Breath sounds	Additional sounds
Pleurisy	Normal	Decreased	Central	Normal	Normal, tender	Decreased	Rub
Pleural Effusion	Bulge	Decreased	Opposite side	Decreased	Basal stony dullness	Decreased	--------
Pneumothorax	Bulge	Decreased	Opposite side	Decreased	Hyper-resonance	Decreased	--------
Hydropneumothorax	Bulge	Decreased	Opposite side	Decreased	Shifting dullness	Decreased	--------
Consolidation	Normal	Decreased	Central	Increased	Dullness	Bronchial	Crepitations
Cavitation	Normal	Decreased	Central	Increased	Dullness	Bronchial	Crepitations
Collapse	Retraction	Decreased	Same side	Decreased	Dullness	Decreased	--------
Fibrosis	Retraction	Decreased	Same side	Decreased	May be patchy dullness	Decreased	Crepitations
Emphysema	Barrel	Decreased	Central	Decreased	Hyperresonance	Decreased Prolonged expiration	Rhonchi, Crepitations
Chronic Bronchitis	Normal	Decreased	Central	Decreased	Normal	Prolonged expiration	Rhonchi

VIP table

DISEASES OF THE PLEURA

1. **Dry pleurisy:**	INFLAMMATION ONLY of the pleura.
2. **Pleural effusion** (Hydro*thorax*):	FLUID in the pleura.
3. **Pneumo***thorax*:	AIR in the pleura.
4. **Hydropneumo***thorax*:	FLUID & AIR in the pleura.
5. **Fibro***thorax*:	FIBROSIS of the pleura.
6. **Mesothelioma** (rare):	TUMOUR of the pleura.

DRY PLEURISY

ETIOLOGY

A) Primary Pleurisy:

1. Idiopathic (probably viral: Coxsakie B or Echo virus): ***the most common cause***
   - Disease: BORNHOLM DISEASE, or PLEURODYNIA.
   - Age: Children & young adults.
   - CP: **Onset** (Acute + fever), **Duration** (few days), **Course** (Recurrent).
2. **Inf**ections: *Viral, Bacterial, TB.*
3. **Inf**lammation: *FMF.*
4. Immunological: *SLE, RA.*
5. Iatrogenic: *INH, Hydralazine, Procainamide.*
6. Irradiation.

7. Renal failure.
8. Rheumatic.

9. **Post**-traumatic.
10. **Post**-cardiotomy syndrome.

11. Myocardial infarction.
12. Malignant infiltration.

B) Secondary Pleurisy: (due to extension from surrounding structures)

1) Lung disease:

- **INFECTIONS** (*TB, Pneumonia, Lung abscess*), **MALIGNANCY** (*Bronchogenic carcinoma*).

2) Mediastinal disease:

- **INFLAMMATION** (*Pericarditis*), **MALIGNANCY** (*Lymphoma*).

3) Subdiaphragmatic:

- **ABSCESS**: *Amoebic liver abscess or Subphrenic abscess.*

4) Chest wall disease:

- **OSTEOMYELITIS**: *Sternum, Ribs or Vertebrae.*

CLINICAL PICTURE

Symptoms

1. Symptoms of the CAUSE.
2. GENERAL symptoms: **e.g.**
   - *Fever, malaise, headache.*
3. LOCAL symptoms:

A) PLEURITIC CHEST PAIN (due to irritation of the parietal pleura)

- **Character & Duration:** Severe Stitching, Continuous.
- **Site & Reference:** ***usually*** *localized* BUT ***may*** *radiate:*
  1. **In inflammation of the central part of the diaphragmatic pleura:** pain radiates to the shoulder through the **phrenic nerve**.
  2. **In inflammation of the outer part of the pleura:** pain radiates to the upper abdomen through the **lower intercostals nerves**. (*this may simulate acute abdomen*).
- **Increases by:** Inspiration, cough & movements of chest wall.
- **Decreases by:** Holding breath & lying on the affected side.
- **Associated symptoms:** May be pain of dry pericarditis.

B) DRY COUGH due to irritation of the pleura.

C) DYSPNEA: *due to ↓↓ of respiratory movements* **or** *to underlying lung disease.*

Signs

1. Signs of the CAUSE.

2. GENERAL signs: **e.g.**
   - *Fever.*

3. LOCAL signs:

Inspection

- Shape: normal.
- Movement: decreased.

Palpation

- **T**rachea: central.
- **T**VF: normal.
- May be palpable pleural rub.

Percussion

- Normal note.
- Tenderness: on the painful site.

Auscultation

- Breath sounds: decreased.
- Additional sounds: " PLEURAL RUB "
  - *It is due to friction between the 2 layers of the rough inflammed pleura.*
    - **Site:** **L**ocalised (at the painful site).
    - **Character:** **L**oud, **L**eathery, **L**ow – pitched.
    - **Increases by:** deep breathing & by pressing the stethoscope against chest the wall.
    - **Disappears:** on holding breath.

Complications

- Pleural effusion.

INVESTIGATIONS

- They have a negligible role in diagnosis, as dry pleurisy is diagnosed essentially:

$$\overbrace{\textit{Pleuritic pain} \quad + \quad \textit{Pleural rub}}^{\textbf{clinically}}$$

- Investigations of the CAUSE.

DIFFERENTIAL DIAGNOSIS

1. From other causes of:	acute chest pain.
2. DD of the causes of:	dry pleurisy.
3. DD of the causes of:	acute abdomen.

TREATMENT

Symptomatic:

- Analgesics & Anti-inflammatory drugs for the pain.

Specific:

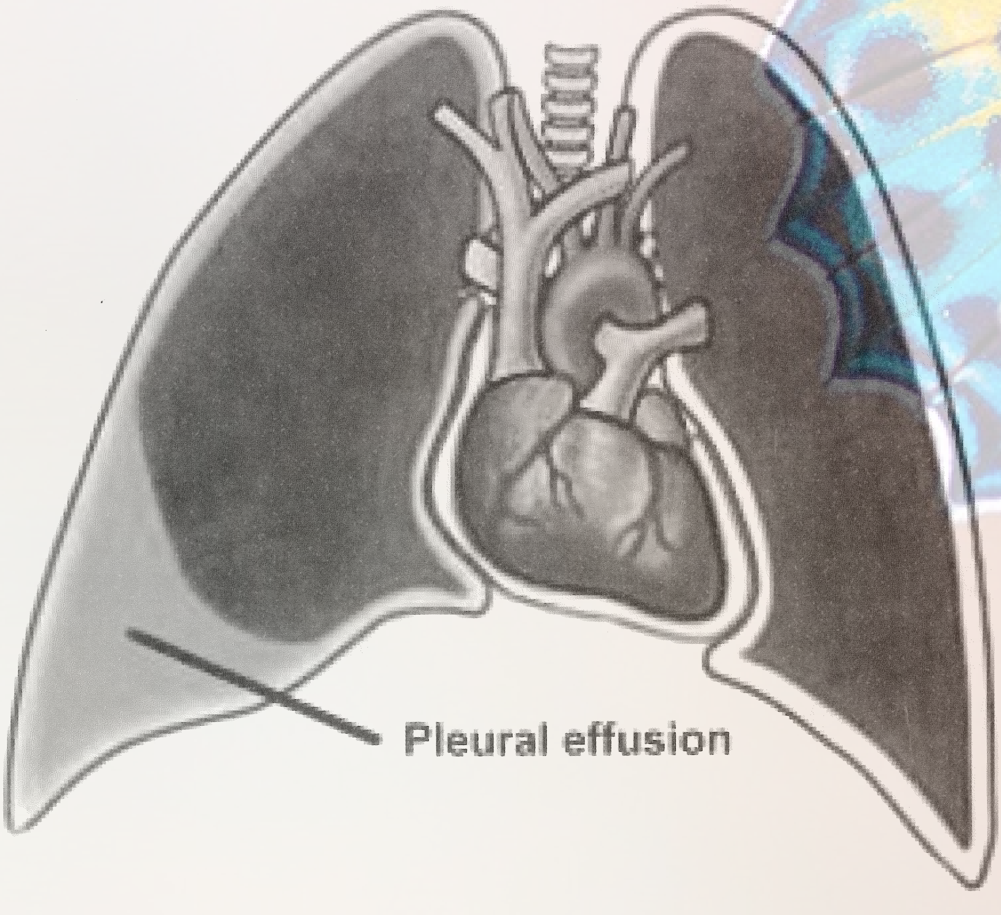
- Treatment of the cause.

PLEURAL EFFUSION

DEFINITION

Abnormal accumulation of: *fluid* in the pleural space:

- *Detected radiologically :* if more than 300 ml.
- *Detected clinically:* if more than 500 ml.



ETIOLOGY

1. Serous effusion (ACCUMULATION OF EXUDATE)

- It occurs in all conditions that cause dry pleurisy especially:
  - **TB:** " *the most common cause of pleural effusion* ".
  - Malignancy.

A) Primary pleural disease.

B) Secondary pleural disease:

1) Lung disease.
2) Mediastinal disease.
3) Subdiaphragmatic.
4) Chest wall disease.

See before

2. Pyothorax (ACCUMULATION OF PUS = EMPYEMA)

- Severe inflammation of the pleura due to:
  Infection by pyogenic organisms, **e.g.** ***Staph.*** & ***Strept.***

3. Hydrothorax (ACCUMULATION OF TRANSUDATE)

- **H**eart failure (RSHF & LSHF), pericardial effusion, CP.
- **H**epatic failure.
- **H**ypoproteinemia, **e.g.** *Nephrotic syndrome*.
- **H**ypothyroidism.
- MEIG'S SYNDROME (*Ovarian tumour*, *ascites*, *right pleural effusion*).
- SVC OBSTRUCTION.

4. **Hemothorax** *(ACCUMULATION OF BLOOD)*

1. Trauma: tapping of effusion & fracture rib.
2. TB.
3. Malignancy: "characteristics of malignant effusion"
    - *Massive, bloody.*
    - *Rapidly re-accumulates after aspiration, contains malignant cells.*
    - *May be: mediastinal shift to the same side (due to underlying collapse).*
4. Hemorrhagic blood diseases.

5. **Chylothorax** *(ACCUMULATION OF LYMPH)*

- Obstruction: of thoracic duct.
- Rupture: of thoracic duct.

CLINICAL PICTURE

Symptoms

1. Symptoms of the cause: **e.g.** **TB toxemia**.

2. Symptoms due to effusion:

    A) Small effusion: *Asymptomatic*.

    B) Large effusion:

    - Dull aching chest pain.
    - Dyspnea.
    - Dry cough.

Signs

1. Signs of the CAUSE: **e.g.** **TB**.

2. LOCAL signs:

Inspection

- Shape: unilateral bulge in large effusion.
- Movement: decreased on the affected side.
- Littin's sign: negative.

Palpation

- Trachea & mediastinum:
  - a) Shift to the opposite side:
    - *Small effusion*: no shift.
    - *Moderate effusion*: shift of the apex to the opposite side.
    - *Large effusion*: shift of the apex & trachea to the opposite side.
  - b) Shift to the same side:
    - *Malignant effusion:* due to underlying lung collapse.
    - *Chronic effusion:* due to pleural fibrosis.
- TVF: decreased on the affected side.

Percussion

- Dullness:
  - *Basal stony dullness*, *with the upper level rising laterally to the axilla.*
  - *Grocco's triangle*.
  - *Garland's triangle*.
- Resonance:
  - *Skodiac resonance*.

Auscultation

- Breath sounds: diminished.
- Additional sounds: PLEURAL RUB may be heard.

COMPLICATIONS

1. Infection: *empyema.*
2. Chronicity: *fibrosis* **or** *permanent lung collapse.*

INVESTIGATIONS

I. IMAGING:

1. CXR:

a) Free effusion:

- **Homogenous opacity obliterating the CP angle & rising laterally:**
  - o *Small effusion*: small opacity with no shift of apex or trachea.
  - o *Moderate effusion*: moderate opacity with shift of apex to opposite side.
  - o *Large effusion*: large opacity with shift of apex & trachea to opposite side.

b) Loculated effusion: ***(especially in empyema)***

- o Fluid may be loculated due to adhesions.
- o Loculated fluid may be either:
  1) *At the periphery of the lung* *(encysted effusion):*
     - Appears as a D – shaped capacity.
  2) *Within the fissures between the lobes* *(interlobar effusion):*
     - Appears as a rounded or oval opacity that may disappear with diuretics: *phantom tumour* or *pseudo tumour*.

2. CT chest:

- o Sensitive in detecting *small amounts* of pleural effusion.
- o May detect *underlying lung disease* **e.g.** malignancy.

II. OTHER INVESTIGATIONS FOR THE CAUSE:

- THORACOCENTESIS: see later.
- Pleural biopsy: ***e.g.*** for TB or malignancy.
- Sputum examination: ***e.g.*** for TB or malignancy.
- Tuberculin test: ***e.g.*** for TB.
- Thoracoscopy: ***e.g.*** for malignancy.

THORACOCENTESIS: (Aspiration of effusion)

- Indications of aspiration:

A] **Diagnostic:** "May be needed for diagnosis of the cause by analysis of the pleural fluid"

a) Features of exudate & transudate effusions:

	TRANSUDATE	EXUDATE
Proteins	< 3 gm %	> 3 gm %
Fluid protein / serum protein	< 0.5	> 0.5
Specific gravity	< 1016	> 1016
LDH	< 200 IU / L	> 200 IU / L
Fluid LDH / serum LDH	< 0.6	> 0.6
Cells (WBCs)	< 1000 / cmm	> 1000 / cmm

NB Characteristics of TB effusion:

1. Exudate: rich in Lymphocytes & RBCs.
2. TB bacilli can be detected by:
   - *Staining:* *ZN stain.*
   - *Culture:* *Lowenstein-Jensen medium or BACTEC.*
   - *Animal Innoculation:* *Guinae pig.*

b) Features of Pyothorax:

- The fluid is ***purulent*** & contains many ***pus cells***.

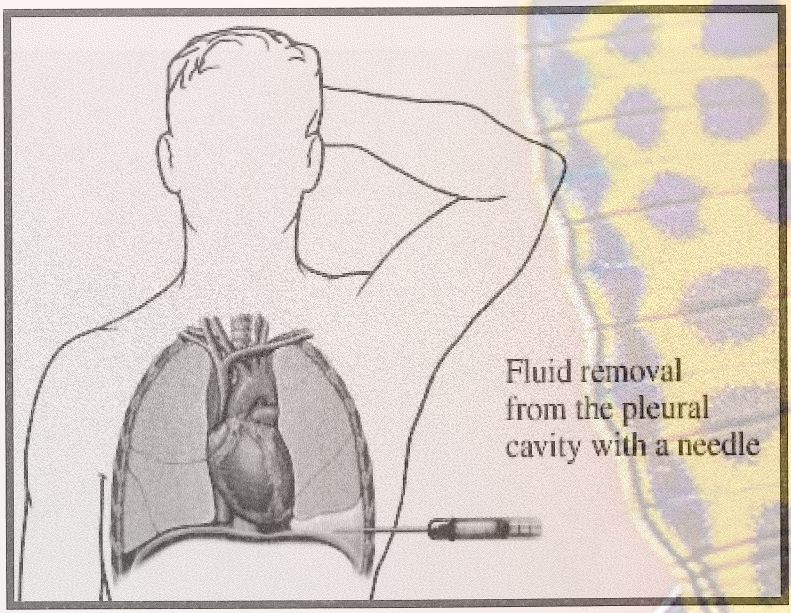
c) Features of Hemothorax:

- The fluid is ***bloody*** & contains many ***RBCs***.

d) Features of Chylothorax:

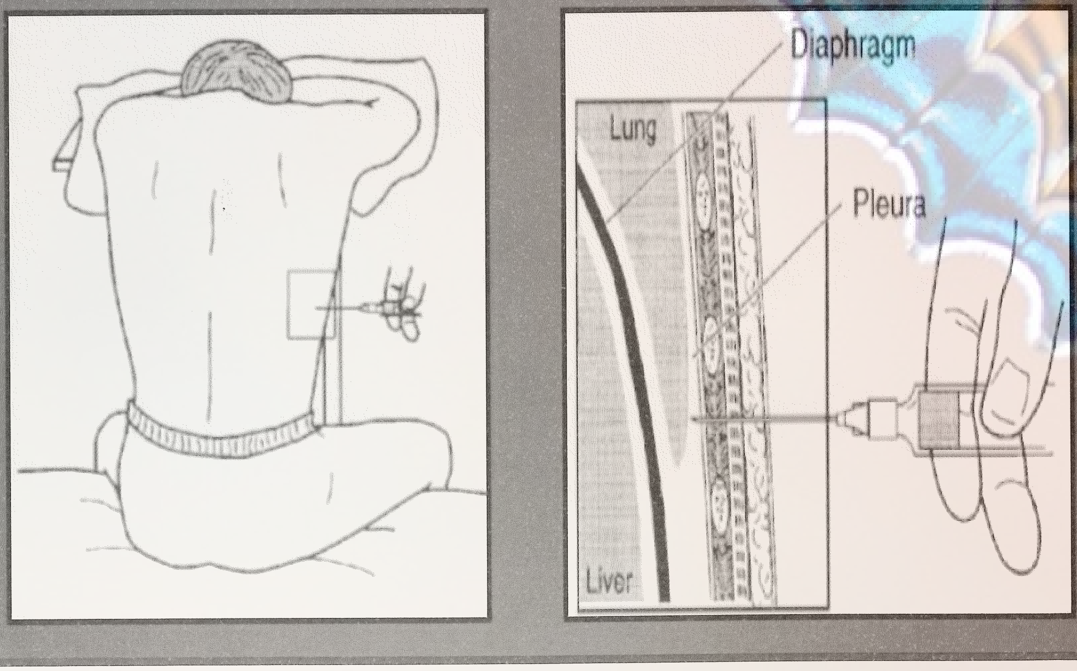
- The fluid is ***milky white*** & contains many ***fat,*** clears on addition of ether & stains orange with: Sudan III.

B] **Therapeutic:** **(see later).**

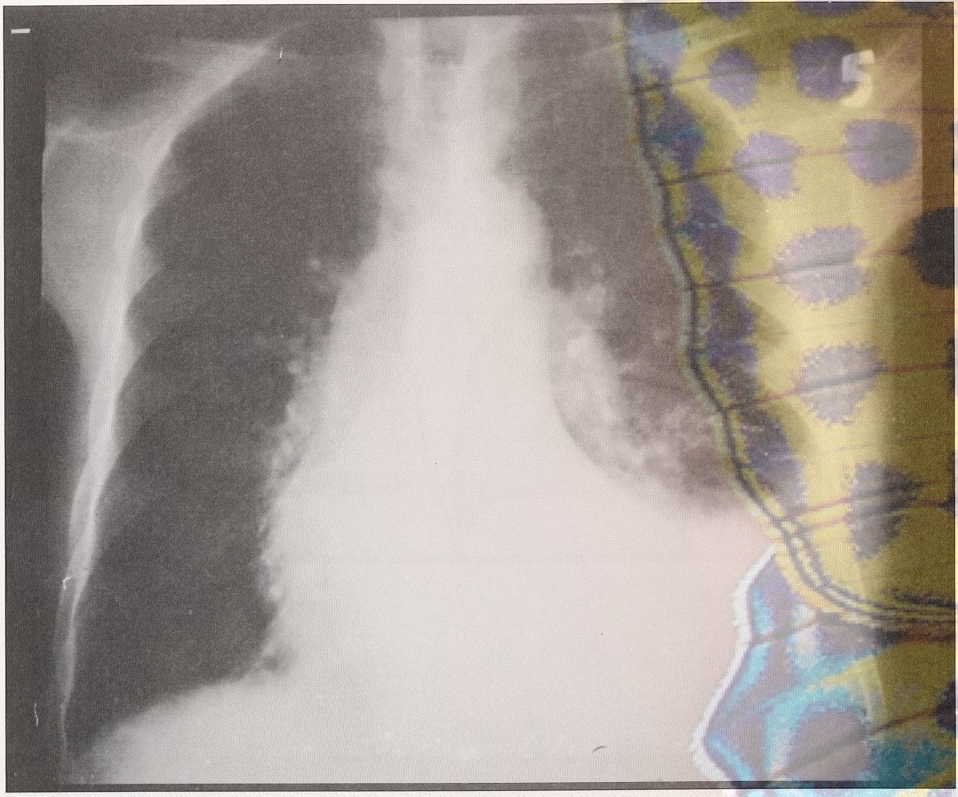


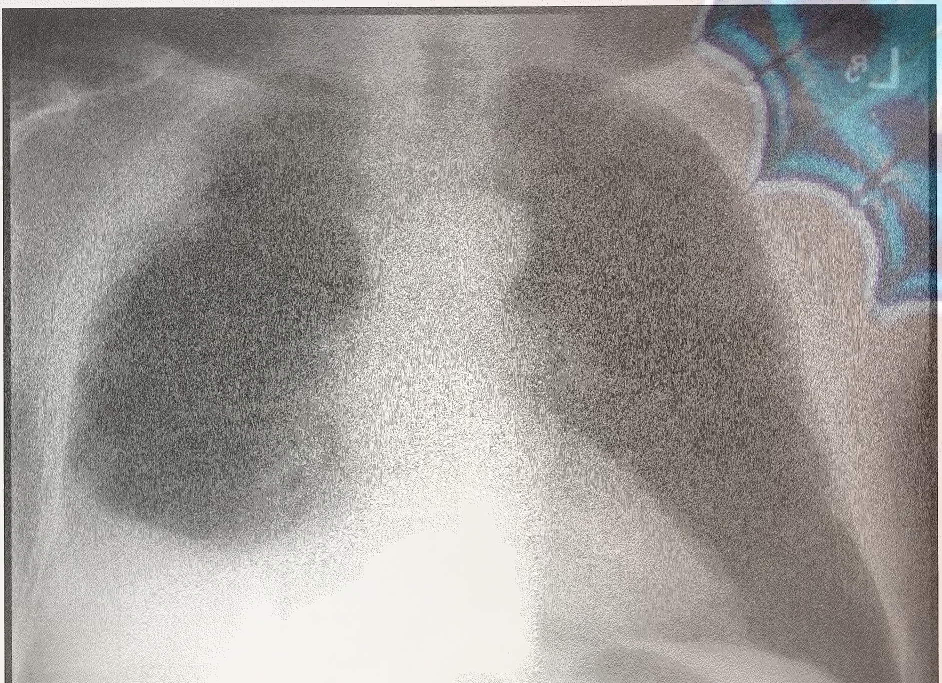
Fluid removal from the pleural cavity with a needle

THORACOCENTESIS


Diaphragm
Lung
Pleura
Liver

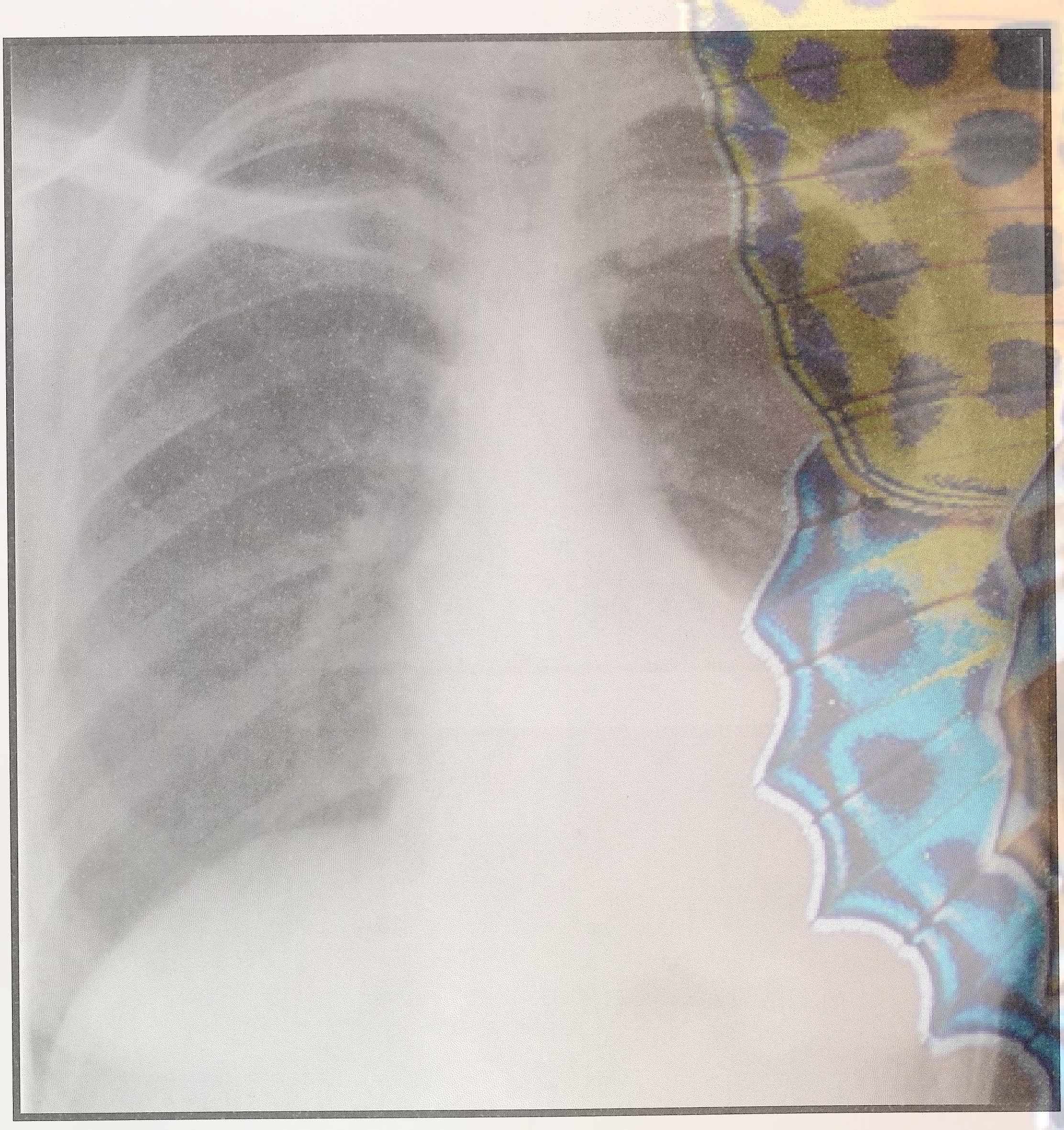
Mild left pleural effusion





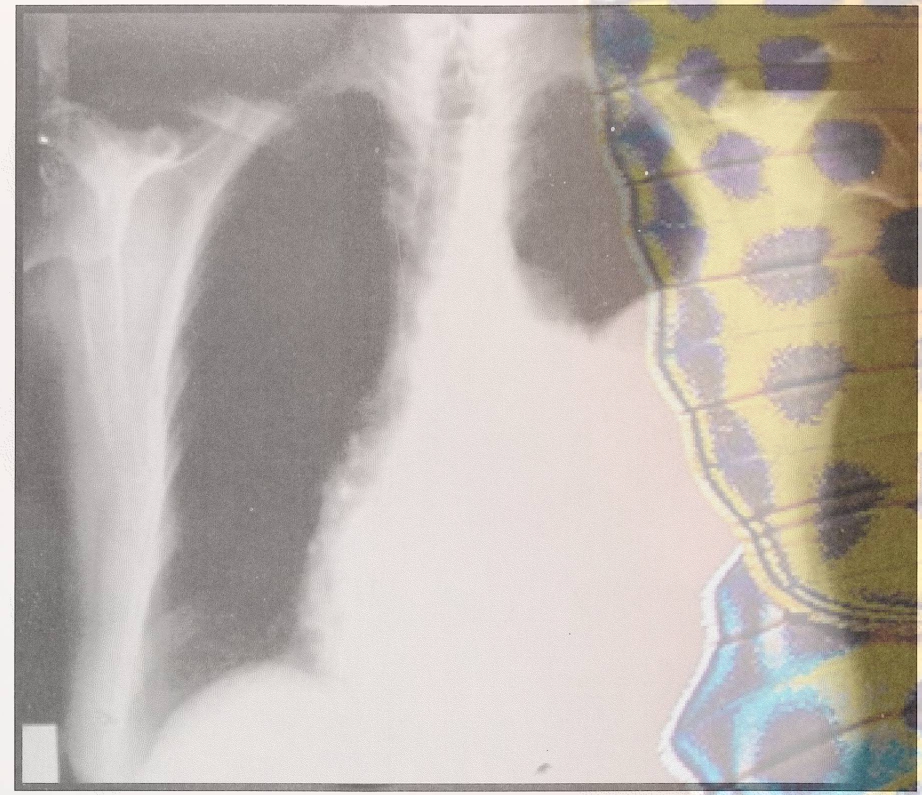
Mild right pleural effusion

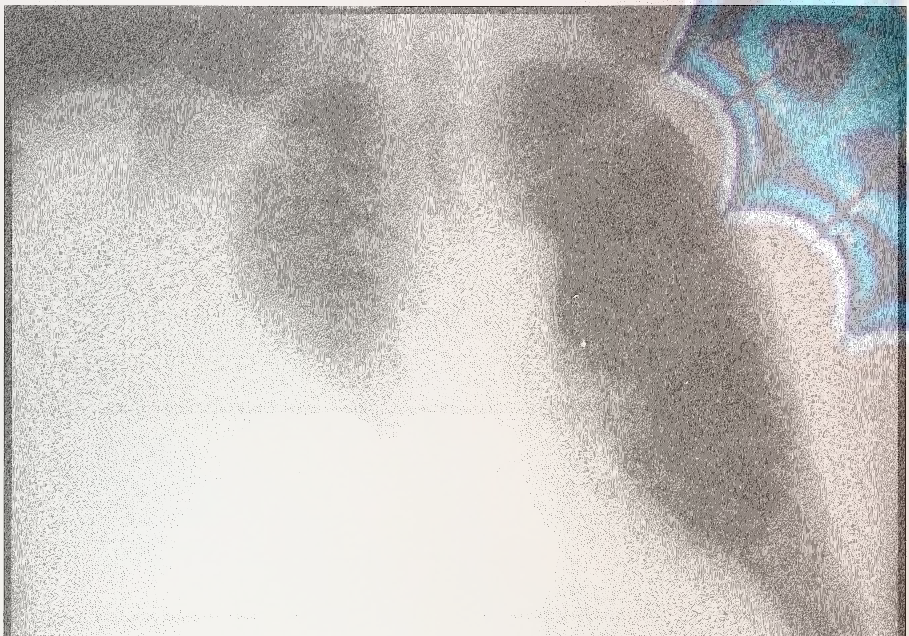
Moderate left pleural effusion



Treat the patient, not the X-ray… ~ **James M. Hunter**

Massive left pleural effusion





Massive right pleural effusion

DIFFERENTIAL DIAGNOSIS

1) **Causes of:** **" syndrome of multiple negatives:**
    - *Pleural effusion, lung collapse, lung fibrosis.*
2) **From basal lung lesions:**
    - *Basal consolidation, collapse, fibrosis.*
3) **From subdiaphragmatic conditions:**
    - *Amoebic liver abscess & subphrenic abscess.*
    - *Tidal percussion may help in differentiation.*
4) **Differential diagnosis of the cause of effusion by:**
    - *Main Features of the cause.*
    - *Nature of pleural fluid.*

TREATMENT

1. Treatment of the cause: e.g.

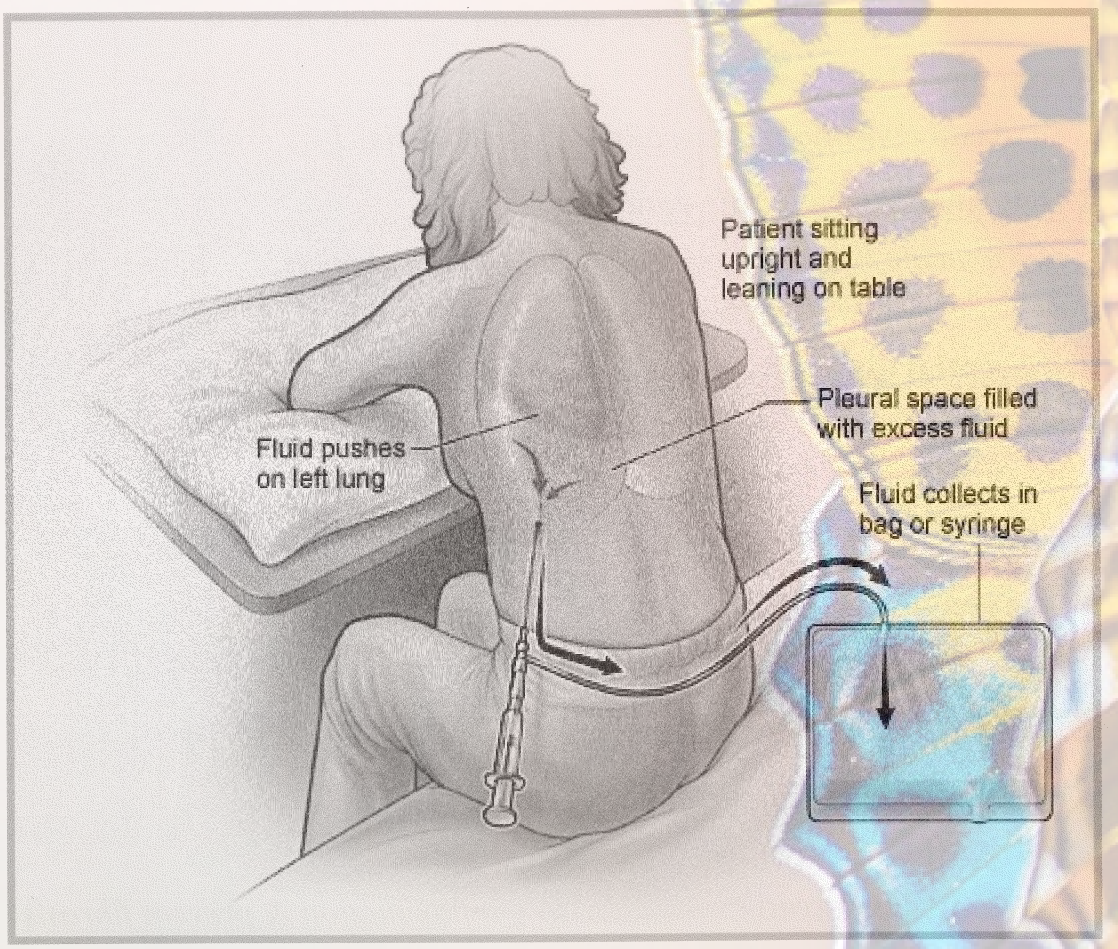
a) *Tuberculous effusion:*
- o Anti – TB drugs.
- o Corticosteroids: *to ↓ inflammation & prevent fibrosis.*

b) *Malignant effusion:*
- o Cytotoxic chemotherapy.
- o Pleural sclerosis: *by intrapleural injection of tetracycline.*

2. THORACOCENTESIS: (Aspiration of effusion)

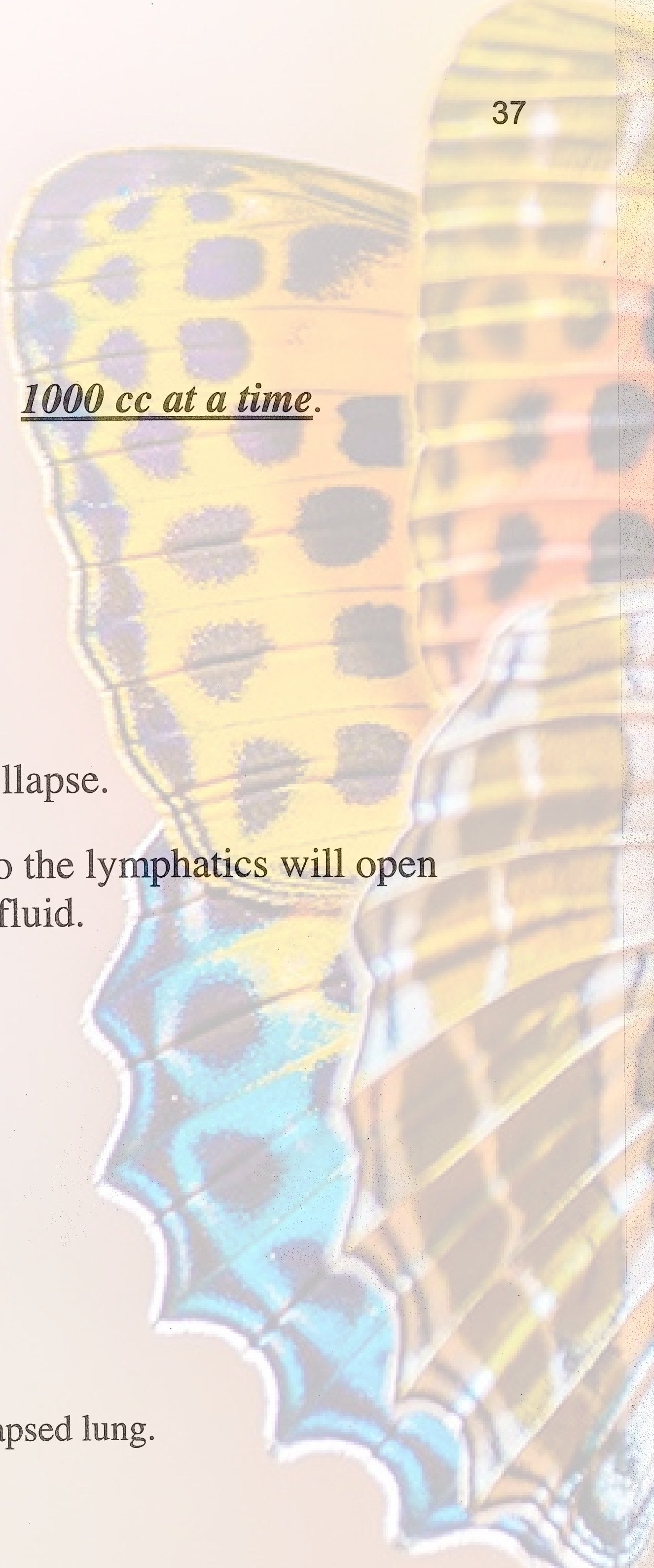
INDICATIONS

1. Massive effusion: { *causing respiratory distress* or *reaching up to the second rib*
2. Resistant effusion: not responding to treatment for ***6 weeks***.
3. Effusion which becomes: ***secondary infected***.


Patient sitting upright and leaning on table
Pleural space filled with excess fluid
Fluid pushes on left lung
Fluid collects in bag or syringe

CONTRAINDICATIONS

1. Bleeding tendency.
2. Uncooperative patient.



PRECAUTIONS

- Aspiration should be done slowly.
- Aspirated amount should not exceed: ***1000 cc at a time***.

BENEFITS

1. Relieves dyspnea.
2. Reduces pleural fibrosis & lung collapse.
3. Reduces the intrapleural pressure, so the lymphatics will open and absorb the remaining amount of fluid.

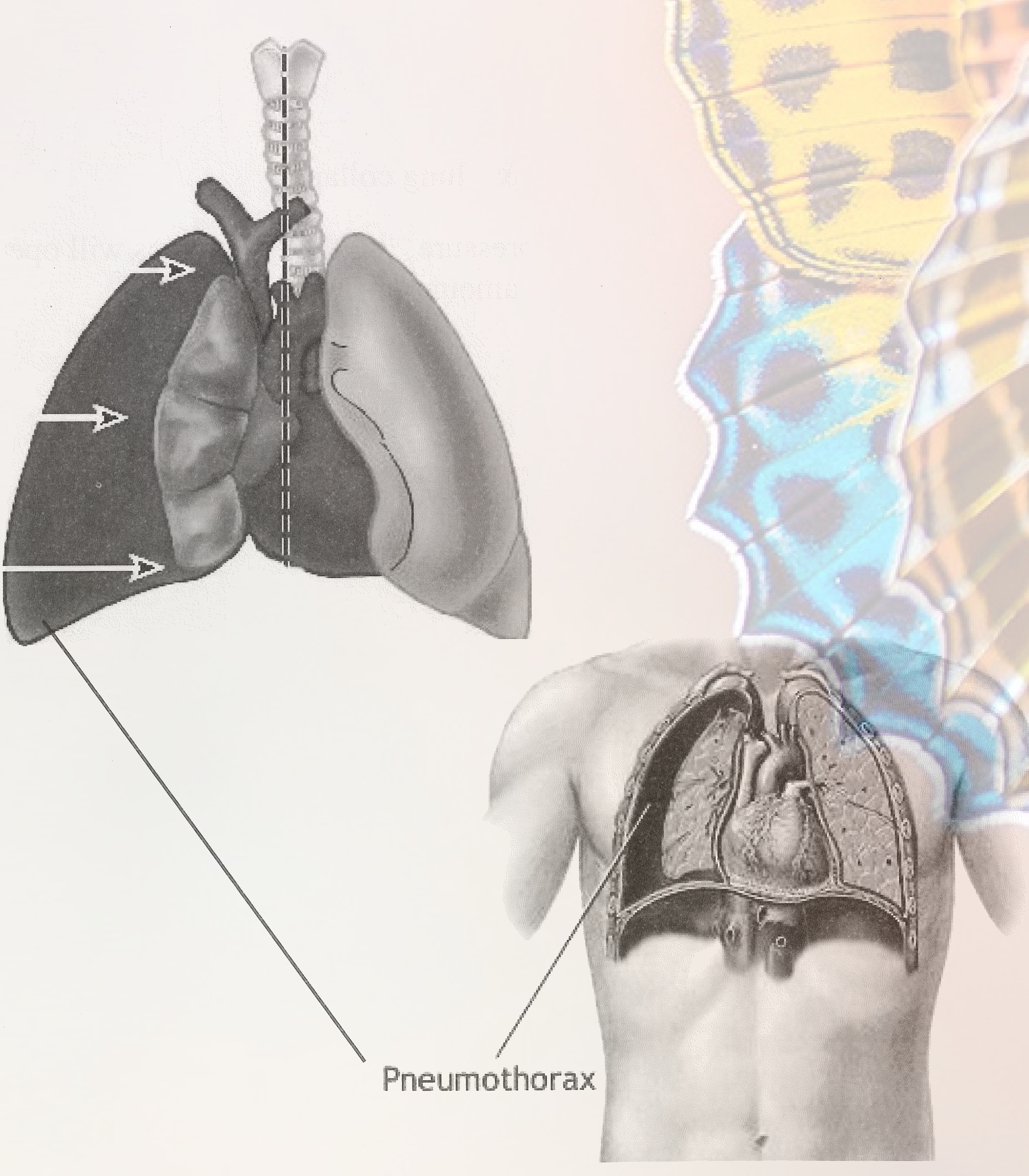
COMPLICATIONS

1. Neurogenic Shock:
   - Due to severe pain.
2. Acute pulmonary oedema:
   - Due to sudden expansion of the collapsed lung.
3. Introduction of:
   - Air → *hydropneumothorax.*
   - Blood → *hemothorax*
   - Infection → *empyema.*
4. Injury:
   - Liver or spleen injury (rare complications).

PNEUMOTHORAX

DEFINITION

Abnormal accumulation of: *air* in the pleural space.


Pneumothorax

ETIOLOGY

1. Spontaneous pneumothorax (non-traumatic):

a. Primary (benign) spontaneous pneumothorax:

- Incidence: most common in males between 20 – 40 years especially smokers.
- Cause: **UNKNOWN,** there is no underlying disease:
  - It may be due to: ***unexplained rupture of a pulmonary bleb*** which is a localised collection of air most commonly in the apical zones.
  - It may be due to: ***a crack in a pleural scar*** resulting from a previous pneumonic patch.
  - It is spontaneously absorbed, but tends to be recurrent.

b. Secondary spontaneous pneumothorax:

- Incidence: related to the underlying disease.
- Cause: **KNOWN,** there is an underlying disease:

1. **LUNG DISEASE:**
   - **Rupture of emphysematous bullae**: **COPD**.
   - Rupture of a TB cavity: TB.
   - Rupture of a lung cyst or abscess.
   - SEVERE ASTHMA.
2. **OESOPHAGEAL DISEASE:** e.g. tumour, (rare).
3. **INFRADIAPHRAGMATIC DISEASE:**
   - Rupture of a subphrenic abscess.

2. Traumatic pneumothorax:

a. Accidental pneumothorax:

- Wounds: penetrating chest injury.
- Invasive measures: e.g. surgery, thoracocentesis, pleural biopsy.
- Endoscopy: *bronchoscopy, oesophagoscopy.*
- MECHANICAL VENTILATION.

b. Artificial pneumothorax:

- Collapse therapy previously used for treatment of TB.

CLINICAL TYPES

1. Closed pneumothorax:

- The tear in the pleura is closed after the occurrence of pneumothorax.
- So the manifestations will: ***subside spontaneously***.

2. Open pneumothorax:

- The tear remains open forming a fistula which communicates: the pleura with the outside atmosphere.
- So the manifestations will: ***remain stationary***.

3. TENSION PNEUMOTHORAX:

- The tear is valve – like allowing air to enter the pleural sac during inspiration but preventing its escape during expiration.
- So the manifestations will be: ***progressive*** *& may be* ***life threatening***.

CLINICAL PICTURE

Symptoms

1. Symptoms of the cause: **e.g. COPD**.

2. Symptoms due to pneumothorax:

A. In closed & open pnemothorax:

- Chest pain: *sudden, severe, tearing, increasing with inspiration.*
- Dyspnea: acute onset.
- Dry cough: acute onset.

B. In tension pneumothorax: "*4 manifestations*"

- Acute chest pain.
- Dyspnea (severe) & cyanosis.
- Acute RVF.
- Shock.

Signs

1. Signs of the CAUSE: **e.g. COPD.**

2. LOCAL signs:

Inspection

- Shape: unilateral bulge in: *tension type*.
- Movement: decreased on the affected side.

Palpation

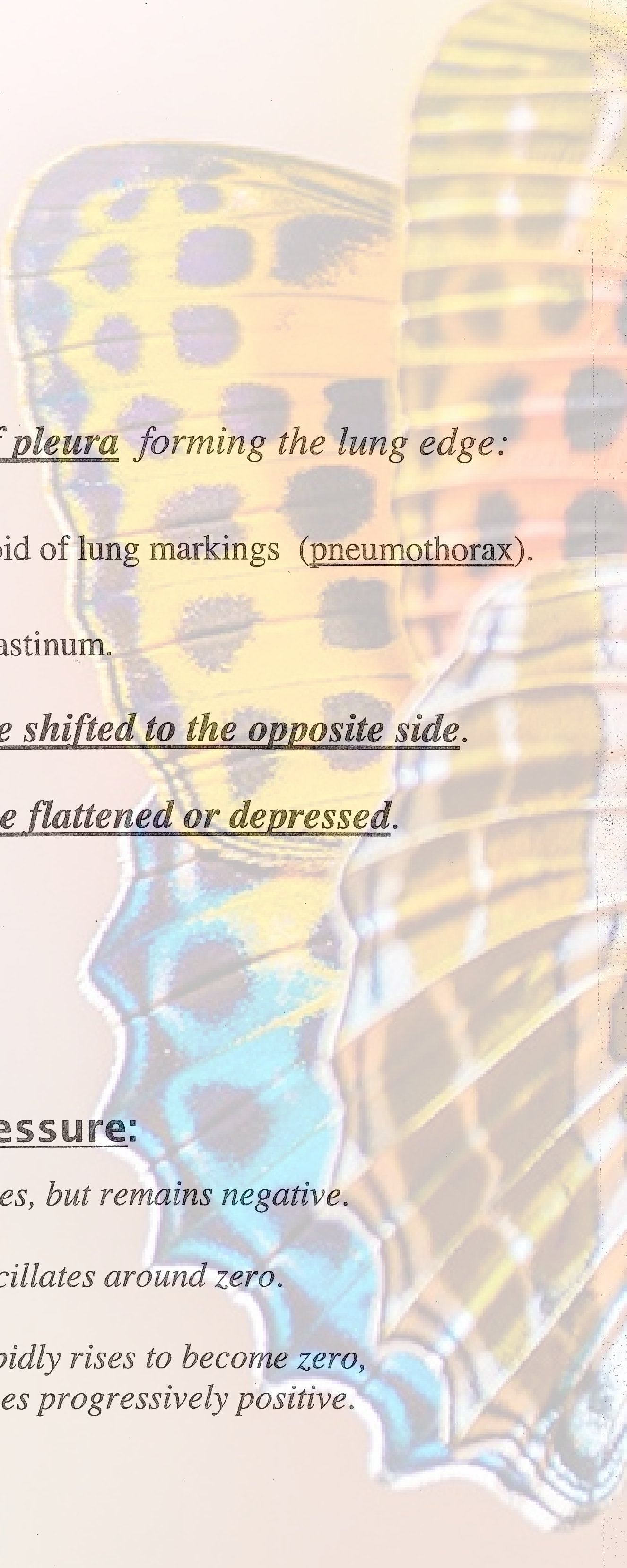
- **T**rachea & mediastinum: shifted to the opposite side in: *tension type*.
- **T**VF: decreased on the affected side.

Percussion

- Hyper-resonance *or* tympanitic resonance.

Auscultation

- Breath sounds: diminished.
- Additional sounds: amphoric breathing in: *open* & *tension types*.



INVESTIGATIONS

1. CXR:

a. *Characteristically, there is* ***the line of pleura*** *forming the lung edge:*

- Outside this line:
  Homogenous jet-black translucency devoid of lung markings (pneumothorax).
- Inside this line:
  The collapsed lung lies close to the mediastinum.

b. *The trachea & mediastinum:* ***may be shifted to the opposite side***.

c. *The copula of the diaphragm:* ***may be flattened or depressed***.

2. Intrapleural manometry:

For recording the intrapleural pressure:

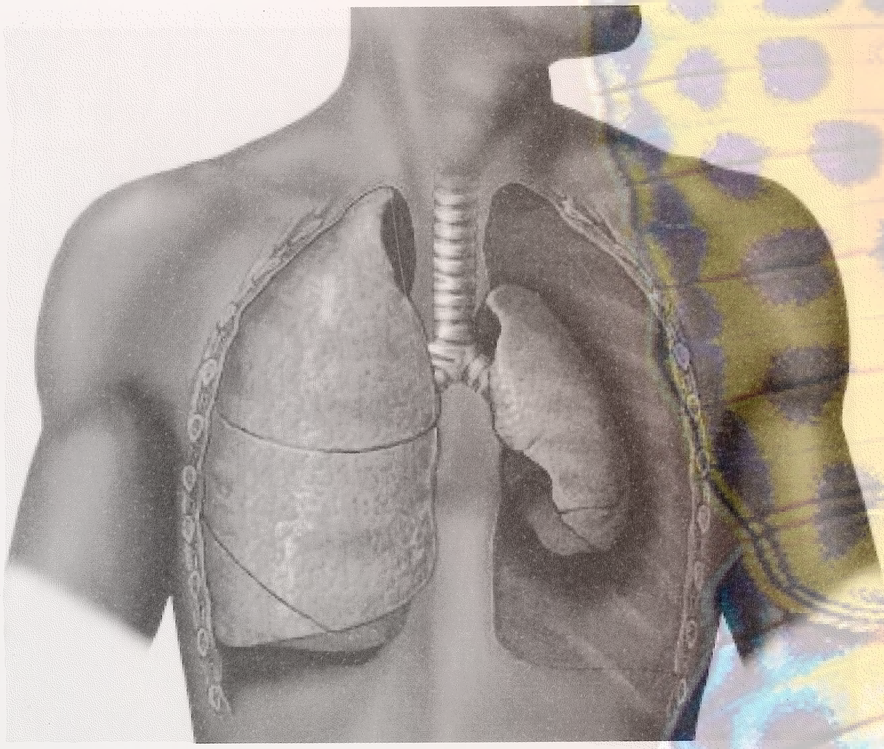
a) In closed pneumothorax: *the pressure rises, but remains negative.*

b) In open pneumothorax: *the pressure oscillates around zero.*

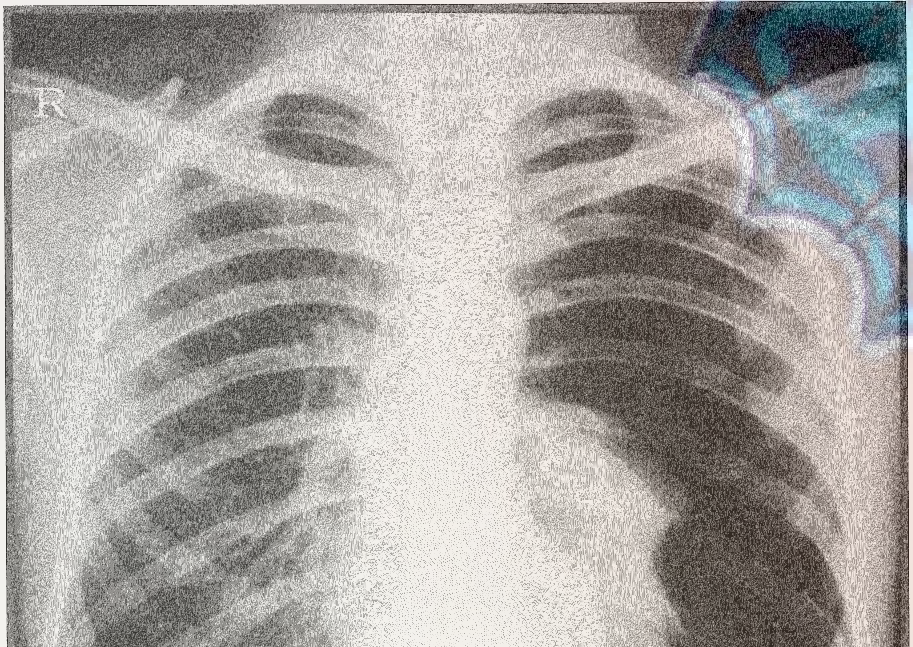
c) In tension pneumothorax: *the pressure rapidly rises to become zero, and then becomes progressively positive.*

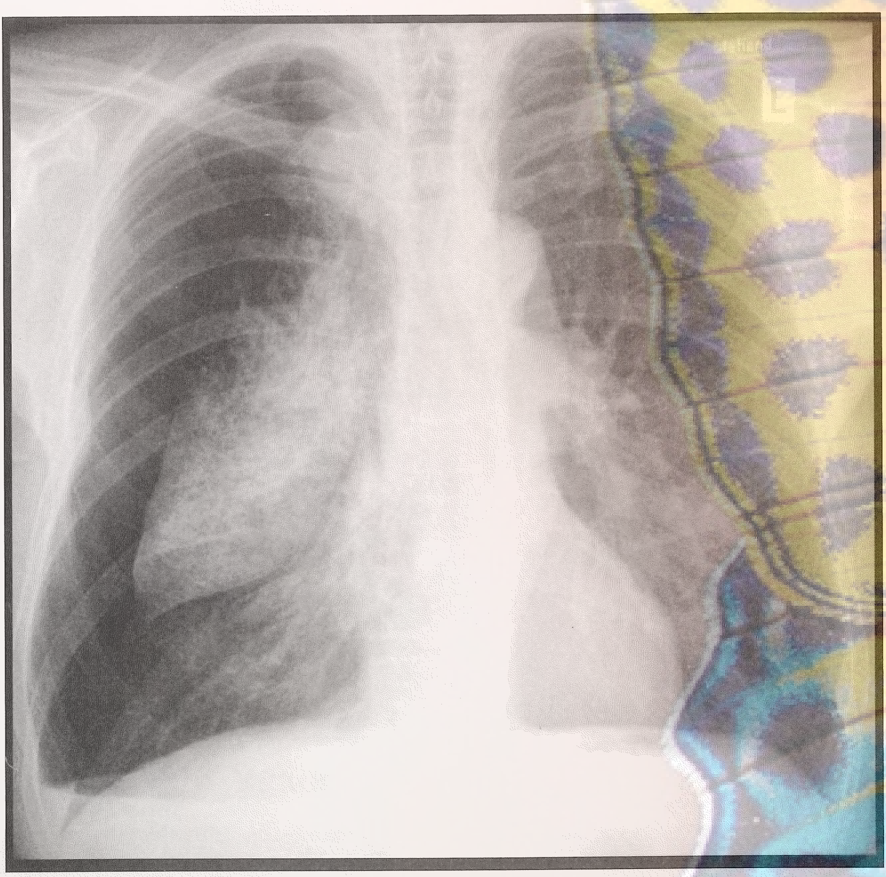
3. ABG:

- Hypoxia.

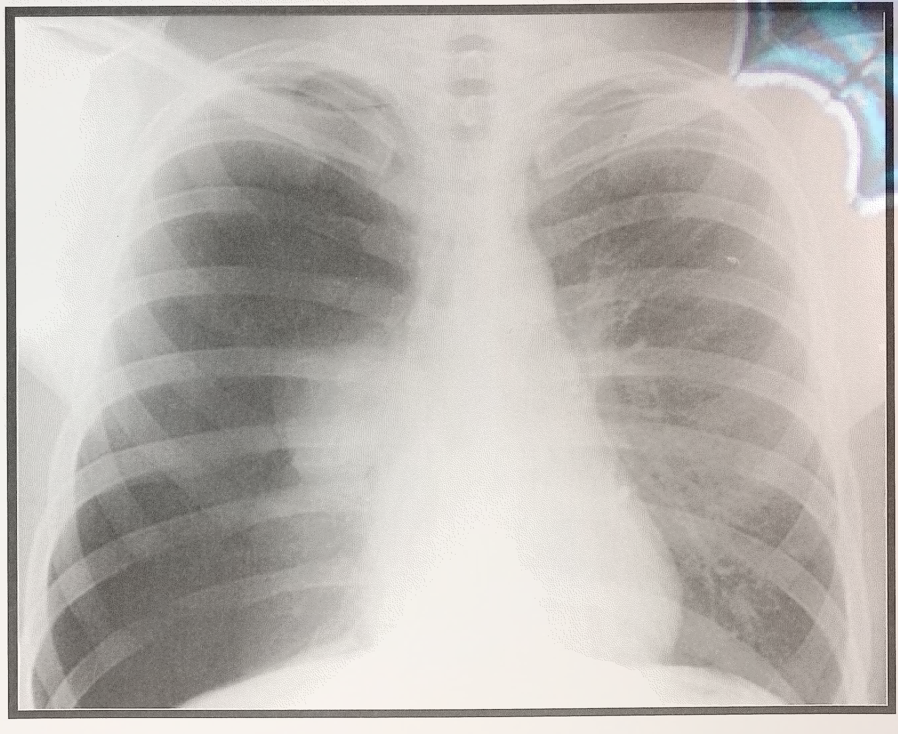


Left pneumothorax





Right pneumothorax



DIFFERENTIAL DIAGNOSIS

1. Differentiation of pneumothorax from:

- Other causes of: *acute chest pain*.
- Other causes of: *acute dyspnea*.
- EMPHYSEMA.

2. Differentiation of tension pneumothorax from:

- Other causes of: ***acute chest pain***, ***dyspnea***, ***acute RVF*** & ***Shock***:
    - Massive myocardial infarction.
    - Massive pulmonary embolism.
    - Massive lung collapse.

3. Differentiation between closed, open & tension pneumothorax:

	Closed	Open	Tension
1. Dyspnea	mild	mild	severe
2. Cyanosis	absent	absent	usually present
3. Shock	absent	absent	usually present
4. Acute HF	absent	absent	usually present
5. Mediastinal shift	absent	absent	usually present
6. Amphoric breathing	absent	present	present
7. Intrapleural manometry	See before		

TREATMENT

1. Conservative treatment:

- It is indicated in: ***primary spontaneous pneumothorax*** with:
    - Mild symptoms.
    - No increase in intrapleural pressure.
    - Small pneumothorax (< 20 % of the hemithoracic volume).
- It consists of: bed rest & analgesics.
- Spontaneous absorption of air occurs allowing re-expansion of the collapsed lung.

2. Under water seal aspiration:

- **It is indicated in:**
  - Severe symptoms.
  - Severe increase in intrapleural pressure.
  - Large pneumothorax (> 20 % of the hemithoracic volume).[*]
  - Tension pneumothorax.

3. Pleural sclerosis: *(production of pleural adhesions)*

- It is indicated in: *recurrent cases*.
- It is done by intrapleural injection of: *tetracycline*.

4. Surgery: *(closure of the tear)*

- **It is indicated in:**
  - Failure of aspiration (lung fails to expand within 10 days).
  - Recurrent pneumothorax.
  - Bilateral pneumothorax.

5. Treatment of tension pneumothorax: *(medical emergency)*

a) Life saving measure:

- Immediate under water seal aspiration: *to ↓ the intrapleural pressure rapidly.*

b) Treatment of the 4 manifestations:

- Immediate relief of *pain:* pethidine 50 mg IM.
- Immediate relief of *dyspnea:* oxygen inhalation.
- Immediate ttt of *heart failure*.
- Immediate ttt of *shock*.

6. Treatment of the cause: in secondary pneumothorax.

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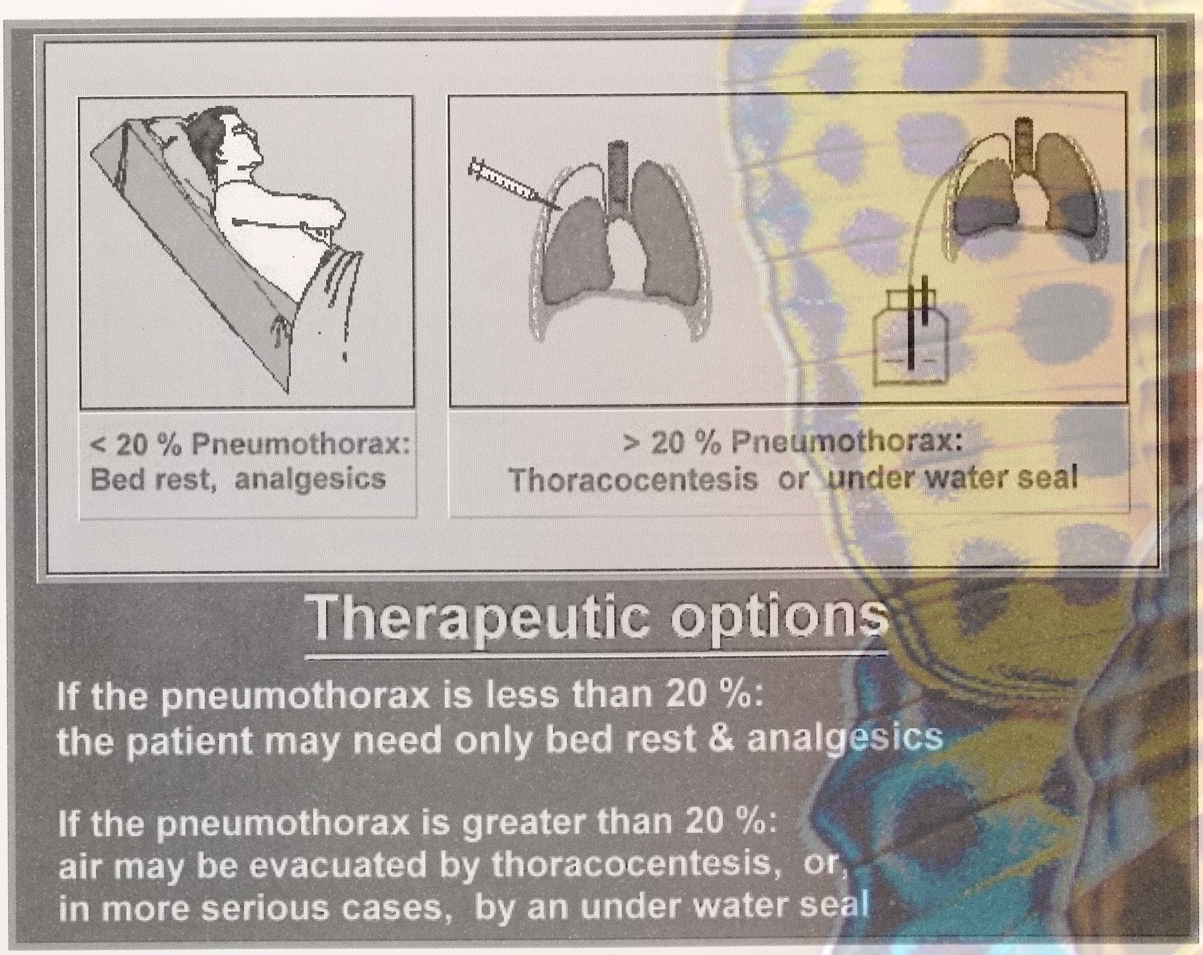
[*] Thoracocentesis may be an alternative in large pneumothorax



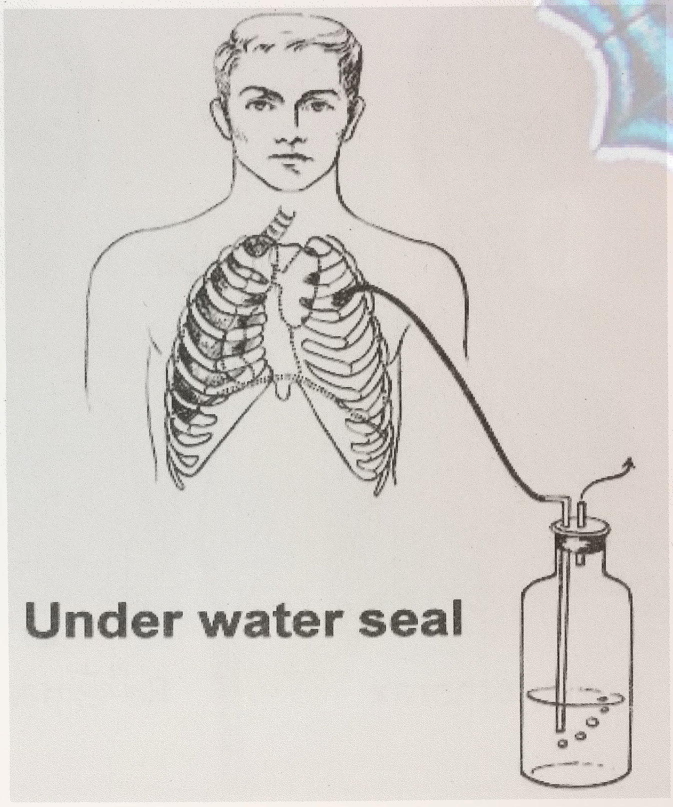
< 20 % Pneumothorax:
Bed rest, analgesics

> 20 % Pneumothorax:
Thoracocentesis or under water seal

Therapeutic options

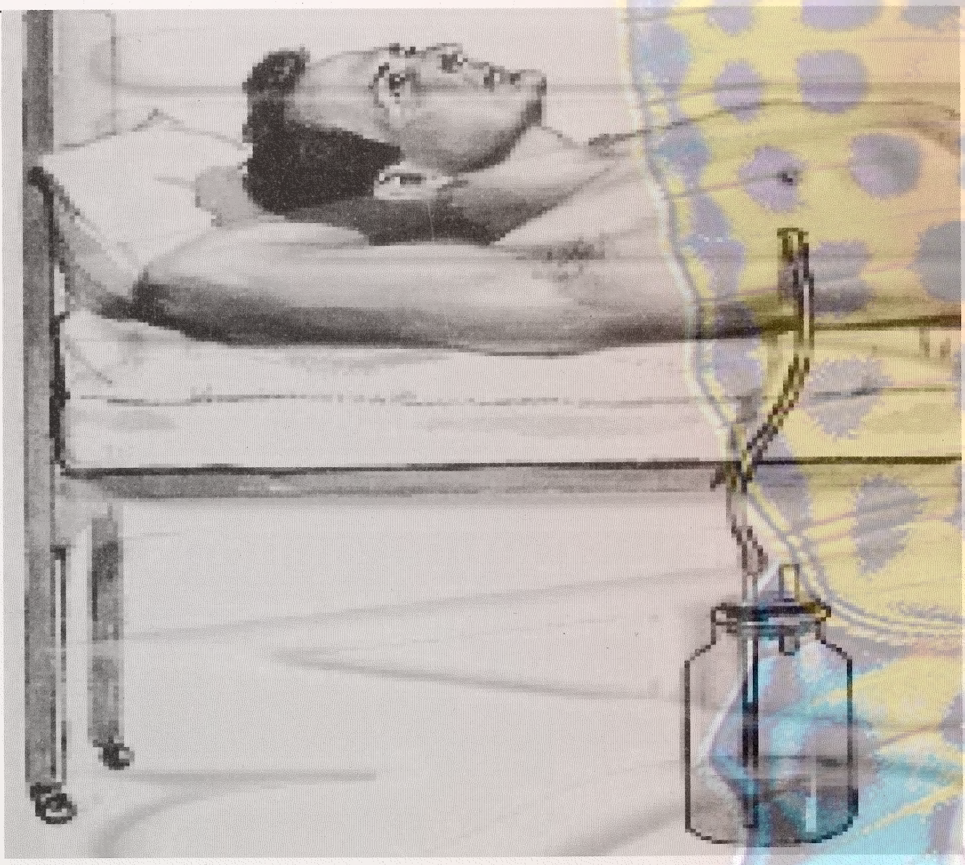
If the pneumothorax is less than 20 %:
the patient may need only bed rest & analgesics

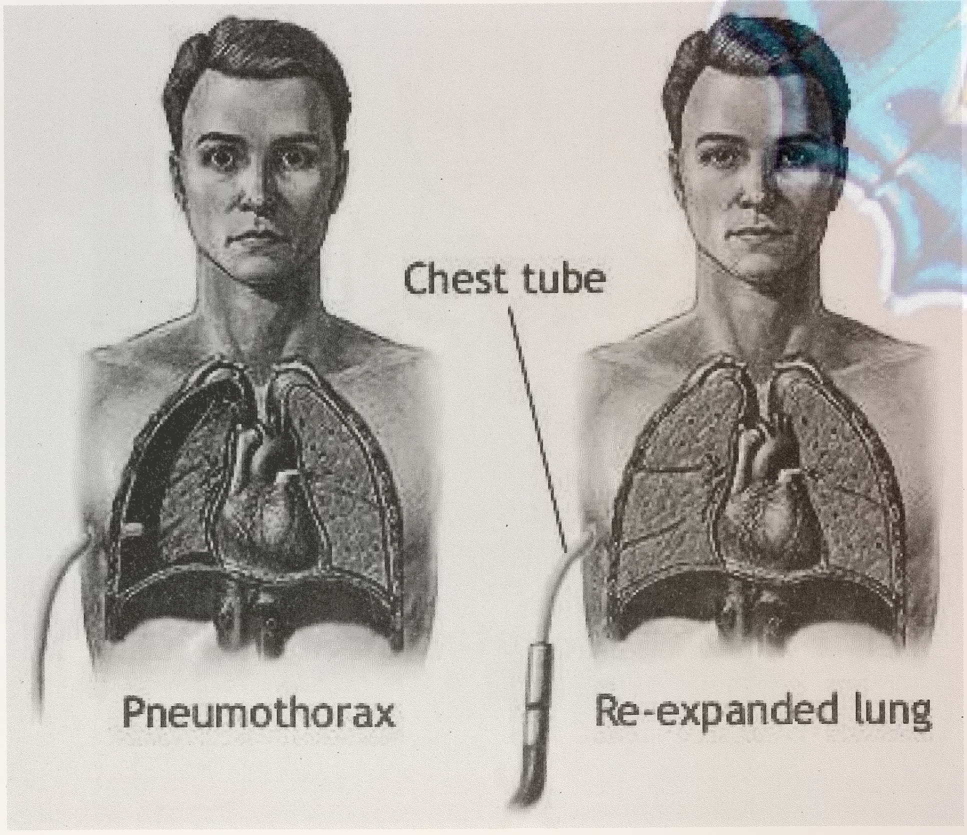
If the pneumothorax is greater than 20 %:
air may be evacuated by thoracocentesis, or,
in more serious cases, by an under water seal



Under water seal

UNDER WATER SEAL




Chest tube
Pneumothorax
Re-expanded lung

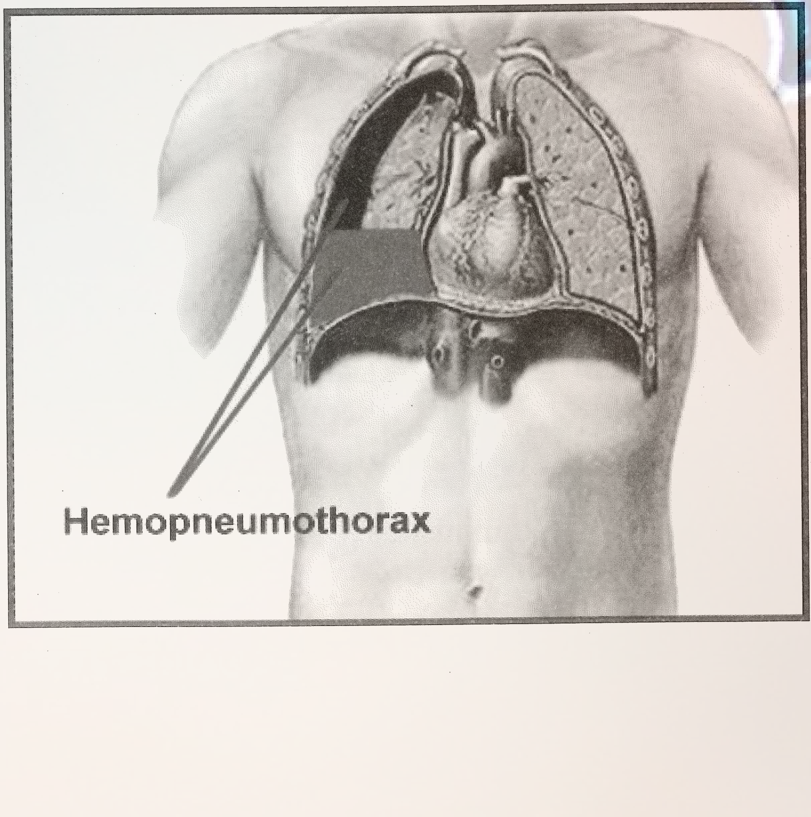
Hydro- , Hemo- & Pyo- PNEUMOTHORAX

DEFINITION

Abnormal accumulation of: *fluid & air* in the pleural space.

ETIOLOGY

1. *Post effusion*:
    - Introduction of air during aspiration of pleural fluid.
    - Empyema opens into a:
        - *Bronchus* (broncho-pleural fistula).
        - *Skin* (pleuro-cutaneous fistula).
2. *Post-pneumothorax*:
    - Hemorrhage or infection complicating under water seal aspiration.
3. *Other causes*:
    - Rupture of: lung abscess or a TB cavity.
    - Rupture of: subphrenic abscess.
    - Penetrating wounds of: chest wall.



CLINICAL PICTURE

Symptoms

- Symptoms of: *the cause* **e.g.** **TB toxemia**.
- Chest symptoms: *chest pain, dyspnea, dry cough.*
- Symptoms of: *suppurative lung syndrome in cases with* **bronchopleural fistula**.

Signs

1. Signs of the CAUSE: **e.g.** **TB**.

2. LOCAL signs:

Inspection

- Shape: unilateral bulge in: *large hydropneumothorax*.
- Movement: decreased on the affected side.

Palpation

- **T**rachea & mediastinum: shifted to the opposite side in: *large hydropneumothorax*.
- **T**VF: decreased on the affected side.

Percussion

- Stony dullness with horizontal upper level (below).
- Hyper-resonance or tympanitic resonance (above).
- Shifting dullness: *is characteristic*.

Auscultation

- Breath sounds: diminished.

INVESTIGATIONS

1) CXR:

a. *Characteristically, there is* ***the line of pleura*** *forming the lung edge:*

- Outside this line:
  - *Below:* homogeneous opacity obliterating the costophrenic angle, and has a horizontal upper level (air-fluid level).
  - *Above:* homogeneous jet-black translucency devoid of lung markings.
- Inside this line:
  - The collapsed lung lies close to the mediastinum.

b. *The trachea & mediastinum:* ***may be shifted to the opposite side.***

c. *The copula of the diaphragm:* ***may be flattened or depressed.***

2) Aspiration of pleural fluid.

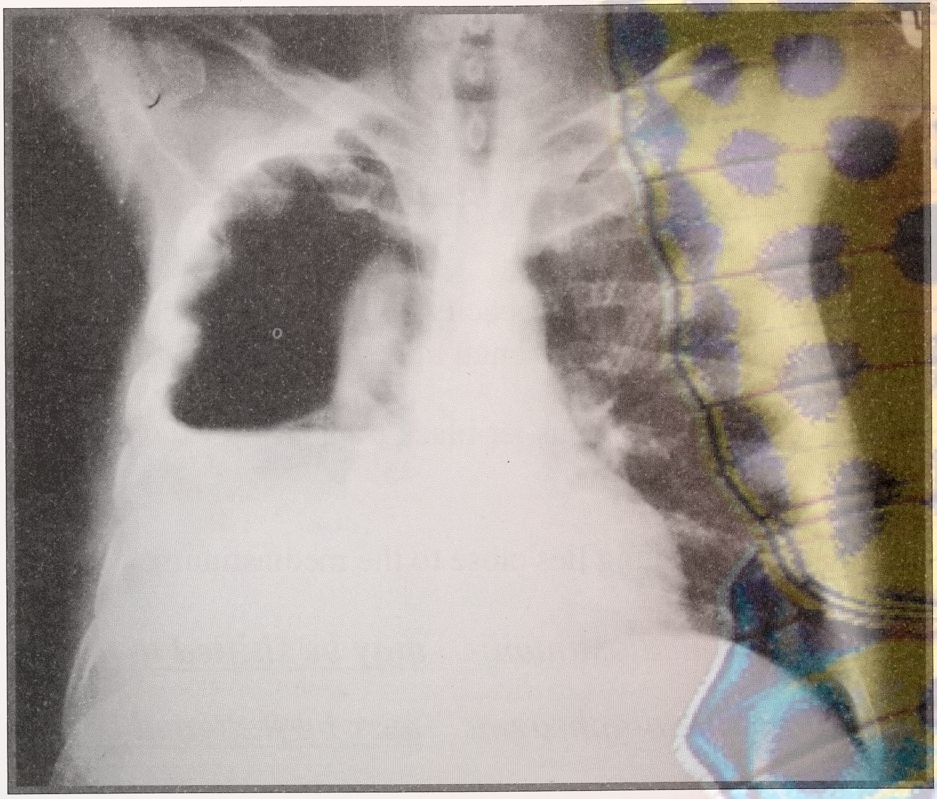
3) Methylene blue test:

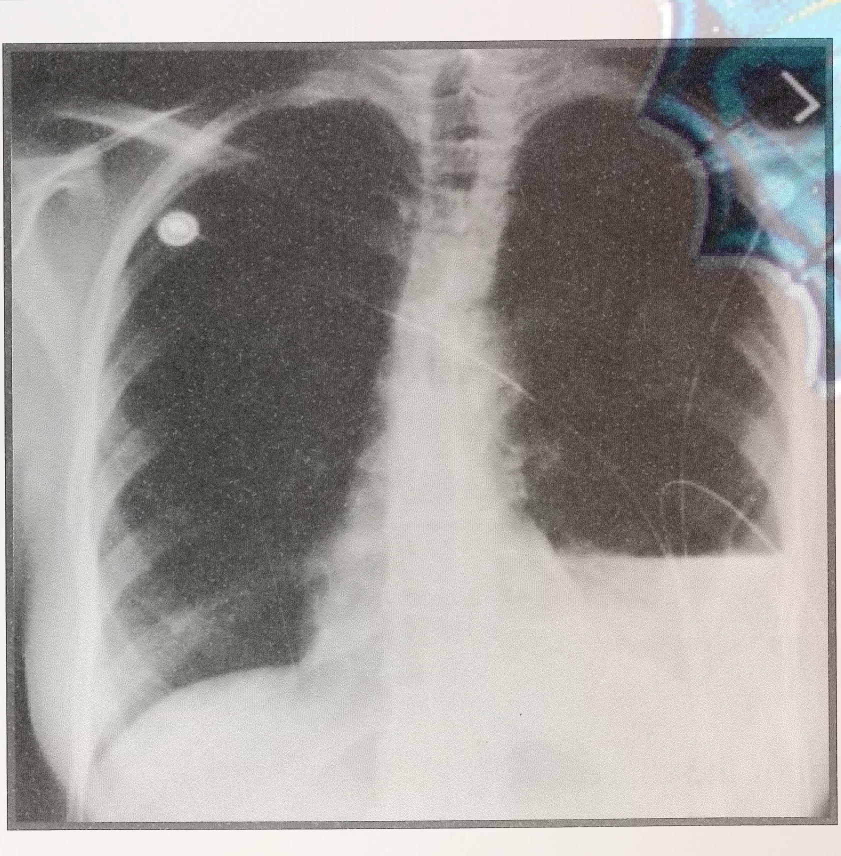
- In the presence of broncho-pleural fistula, Methylene blue injected in the pleura will appear in the sputum.

TREATMENT

1. Treatment of the cause.
2. **Aspiration of fluid & air using an *under water seal.***

Right hydropneumothorax





Left hydropneumothorax

PNEUMONIA

DEFINITION

- INFLAMMATION of the lung: (usually acute)
  - Terminal airways.
  - Alveoli.
  - Interstitial tissue.
- It may be caused by: ***infective*** & ***non – infective*** factors.

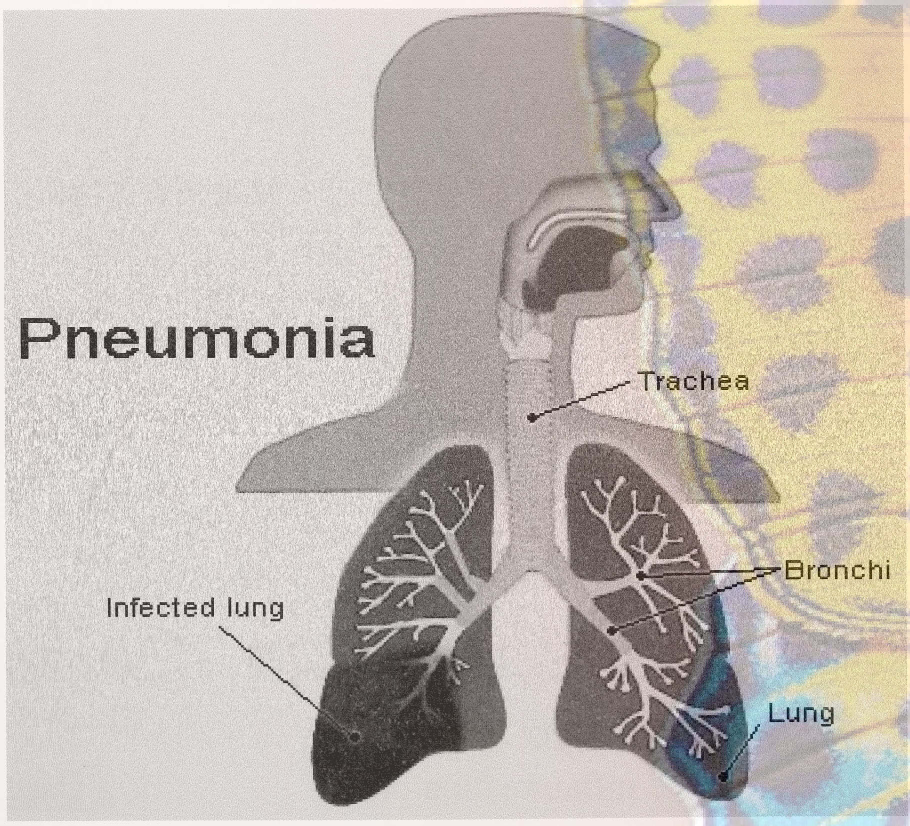
CLASSIFICATION

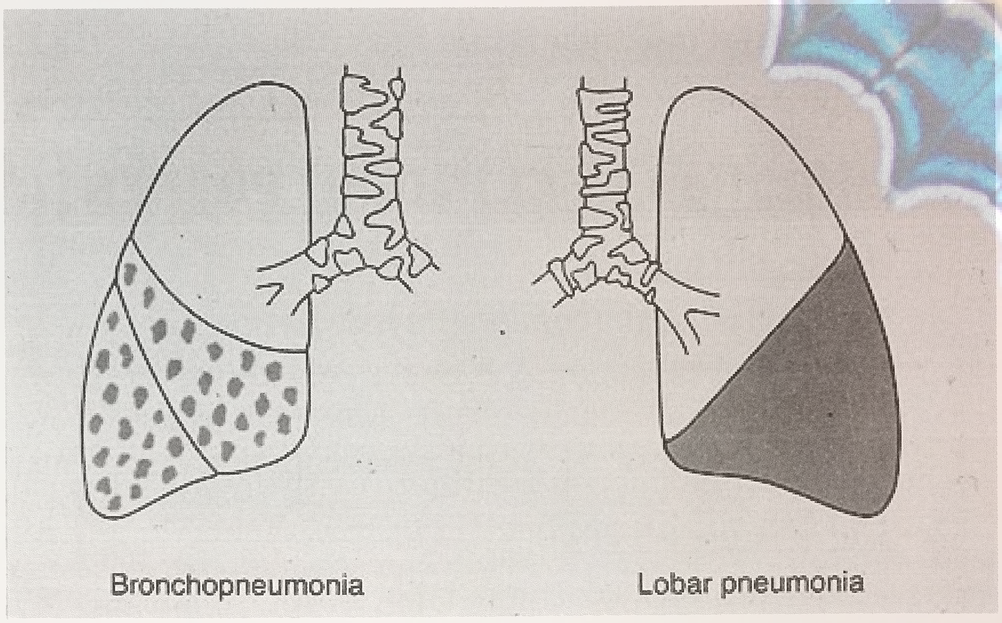
A. Classification by LOCATION (Anatomical):

1. **Lobar pneumonia:**
   - Localised inflammation of: a ***single entire lobe***.
2. **Bronchopneumonia:**
   - Diffuse bilateral patchy inflammation of: ***multiple lobules***.
     (affecting mainly: the alveoli adjacent to the bronchioles).
3. **Interstitial pneumonia:**
   - Inflammation of: ***the interstitial tissue inbetween the alveoli***.

B. Classification by CLINICAL PRESENTATION:

1. **Acute pneumonia:**
   - It is the usual (common) type:
     - Onset: acute.
     - Duration: short (less than *3 weeks*).
     - Cause: the usual common organisms (see later).
2. **Chronic pneumonia:**
   - It is the unusual (rare) type:
     - Onset: gradual.
     - Duration: long (more than *3 weeks*).
     - Cause: mycobacteria, fungi, non – infective.


Pneumonia
Trachea
Bronchi
Infected lung
Lung


Bronchopneumonia
Lobar pneumonia

C. Classification by ETIOLOGY:

I. INFECTIVE: [Most common]

1. The infecting organism:

- BACTERIA:
  - Gm + ve cocci: *Strept. pneumoniae, Staph. aureus. St. pyogenes.*
  - Gm - ve bacilli: *H. influenzae, Klebsiella, Pseudomonas, Legionella.*
  - Anaerobes.
  - Mycobacterium TB.
- Viruses:
  - Influenza virus, adenovirus, varicella, RSV, CMV.
- Mycoplasma.
- Chlamydia.
- Rickettsia.

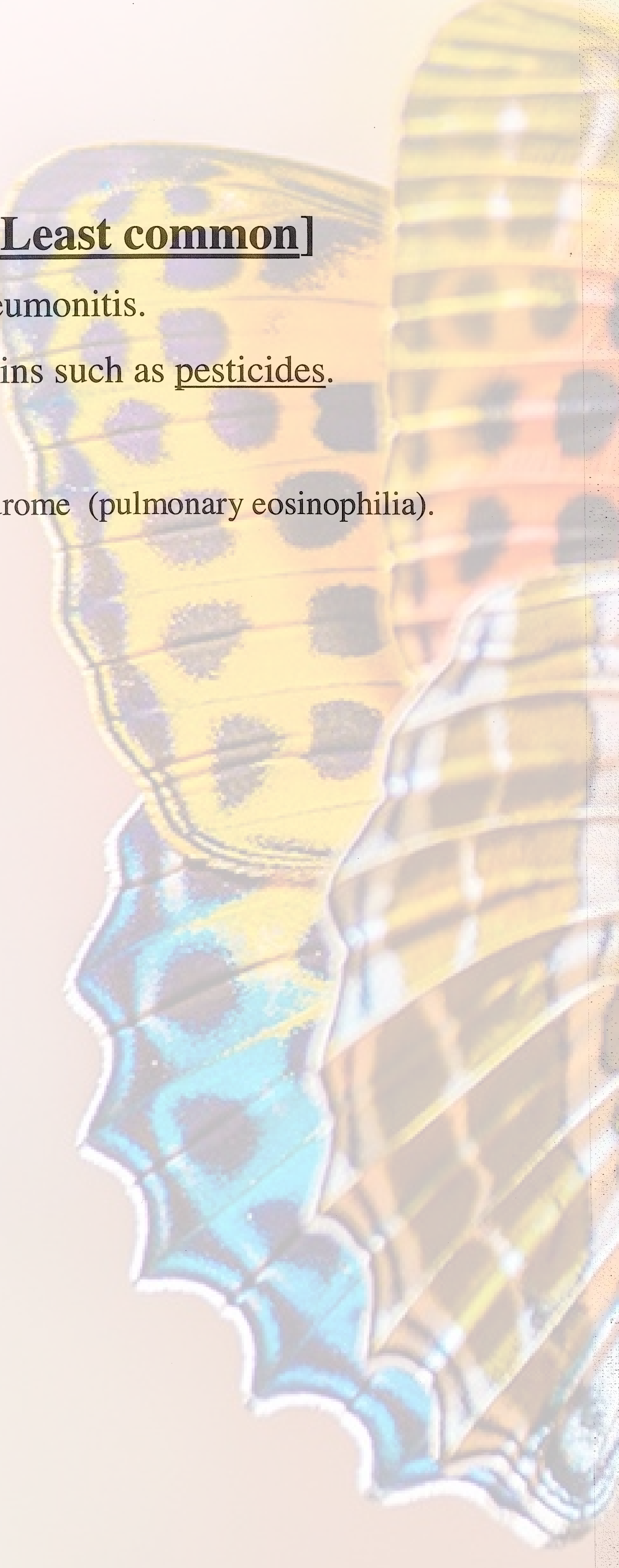
- Fungi: **e.g.** *Histoplasma, Candida.*
- Protozoa: **e.g.** *Pneumocystis carinii.*
- Parasites: **e.g.** *Filaria, Toxoplasma, Ascaris.*

2. The mode of infection:

- Community – acquired pneumonia:
  - Strept. pneumoniae (pneumococcal pneumonia): ***most common***.
  - Others: *H. influenzae, Legionella, Mycoplasma, Chlamydia, Viruses.*
- Hospital – acquired (Nosocomial) pneumonia:
  - Risk factors: Mechanical ventilators (VAP), IV cannulae.
  - Organisms:
    - Staph. aureus: ***most common***.
    - Others: *Klebsiella, Pseudomonas, Anaerobes.*
- Aspiration pneumonia:
  - Risk factors: Stroke, Bulbar palsies, DCL, Anaesthesia, *GORD*.
  - Organisms:
    - Anaerobes: ***most common***.
    - Others: *Strept. pneumoniae, Staph. aureus, H. influenzae, Klebsiella.*
- Pneumonia in the immunocompromised patient:
  - Risk factors: HIV, BM ↓, advanced malignancy, immunosupp. drugs.
  - Organisms:
    - Pneumocystis carinii: ***most common***.
    - Others: *Fungi, CMV, TB.*

II. NON – INFECTIVE: [Least common]

- *Physical:* radiation pneumonitis.
- *Chemical:* chemical toxins such as pesticides.
- *Immunological:* SLE.
- *Allergic:* Loffler’s syndrome (pulmonary eosinophilia).



LOBAR PNEUMONIA

CAUSATIVE ORGANISM

- It is usually caused by: ***Strept. pneumoniae***.

PREDISPOSING FACTORS

- AGE: *middle age*.
- PRECEDING INFECTION: *upper respiratory tract viral infection.*
- ENVIRONMENT: *cold weather*, *overcrowding*.
- ↓↓ IMMUNITY: *malnutrition*, *hyposplenism*.

PATHOLOGY

A) LOCATION:

- Unilateral, usually involving one whole lobe.

B) LESION:

- ***Consolidation*** & ***acute pleurisy***.

- The disease passes through 3 stages over 7 days:

a) Stage of congestion:

- The alveoli are congested with: *minimal exudate*.
- Air diminishes in the alveoli.

b) Stage of hepatization:

- The alveoli are filled with: *RBCs*, *WBCs* *fibrin*, *thick exudate*.

c) Stage of resolution:

- The exudate is **liquefied** by enzymatic activity & becomes *copious* & *watery*.
- The exudate is then **cleared** by:
  - Macrophages: through the blood and lymph vessels, **or:**
  - Cough mechanism.

CLINICAL PICTURE

Symptoms (**Typical pneumonia**)

A. General symptoms

- *Fever:*
  - Acute onset, high, associated with headache, malaise & rigors.
- *Associated manifestations:*
  - Somnolence & confusion: *especially in the elderly.*
  - Headache & convulsions: *especially in children.*

B. Chest symptoms

- *Dyspnea.*
- *Pain:*
  1. Pleuritic stitching.
  2. Upper abdominal pain & rigidity may occur in case of inflammation of the outer pleura & may simulate ***acute abdomen***.
- *Cough:*
  1. Dry: *in the early stage*.
  2. Productive: *later:*
     - With rusty sputum: during hepatization. *(may be yellowish or greenish, with blood streaking).*
     - With watery sputum: during resolution.

Signs

A. General signs

- *Fever:*
  - High continuous fever (40° C), lasts for about 7 days.
  - Resolves abruptly ***(by crisis)***.
- *RR:*
  - Tachypnea (40 / min).
- *Face:*
  - Pale & toxic.
- *Lips:*
  - Herpes simplex eruptions.

B. Chest signs (*Signs of consolidation & pleurisy*)

Inspection

- Shape: normal shape of the chest.
- Movements: limited on the affected side.

Palpation

- **T**rachea & mediastinum: central.
- **T**VF: increased.

Percussion

- Dullness: on the affected area (lobe), due to ***consolidation***.
- Tenderness: on the affected area (lobe), due to ***pleurisy***.

Auscultation

- Breath sound: bronchial breathing.
- Additional sounds: crepitations:
  - **Fine:** **early stage**
  - **Medium sized:** **during hepatization**
  - **Coarse:** **during resolution**
- Pleural rub: is usually present.

COMPLICATIONS

A. General complications

- *Toxic myocarditis:* may lead to HF.
- *Bacteremia:* meningitis, peritonitis or suppurative arthritis.
- *Septicemia:* in severe cases may cause DIC, ARDS.

B. Chest complications

- *Unresolving pneumonia* (persisting more than 2 weeks).
- *Lung abscess*.
- *PLEURA:* *Pleural effusion,* *Empyema.*

UNRESOLVING PNEUMONIA

- **DEFINITION:** Pneumonia that persists more than *2 weeks*.
- **CAUSES:**
  1. Inadequate treatment.
  2. Underlying lung disease **e.g.** **b**ronchiectasis & **b**ronchial carcinoma.
  3. TB pneumonia.
  4. Decreased immunity:
     - **D**iabetes mellitus.
     - **D**rugs: *Corticosteroids* & *other immunosuppressives*.
     - **A**IDS.
     - **M**alignancies, *especially lymphomas*.
     - **M**alnutrition.

INVESTIGATIONS

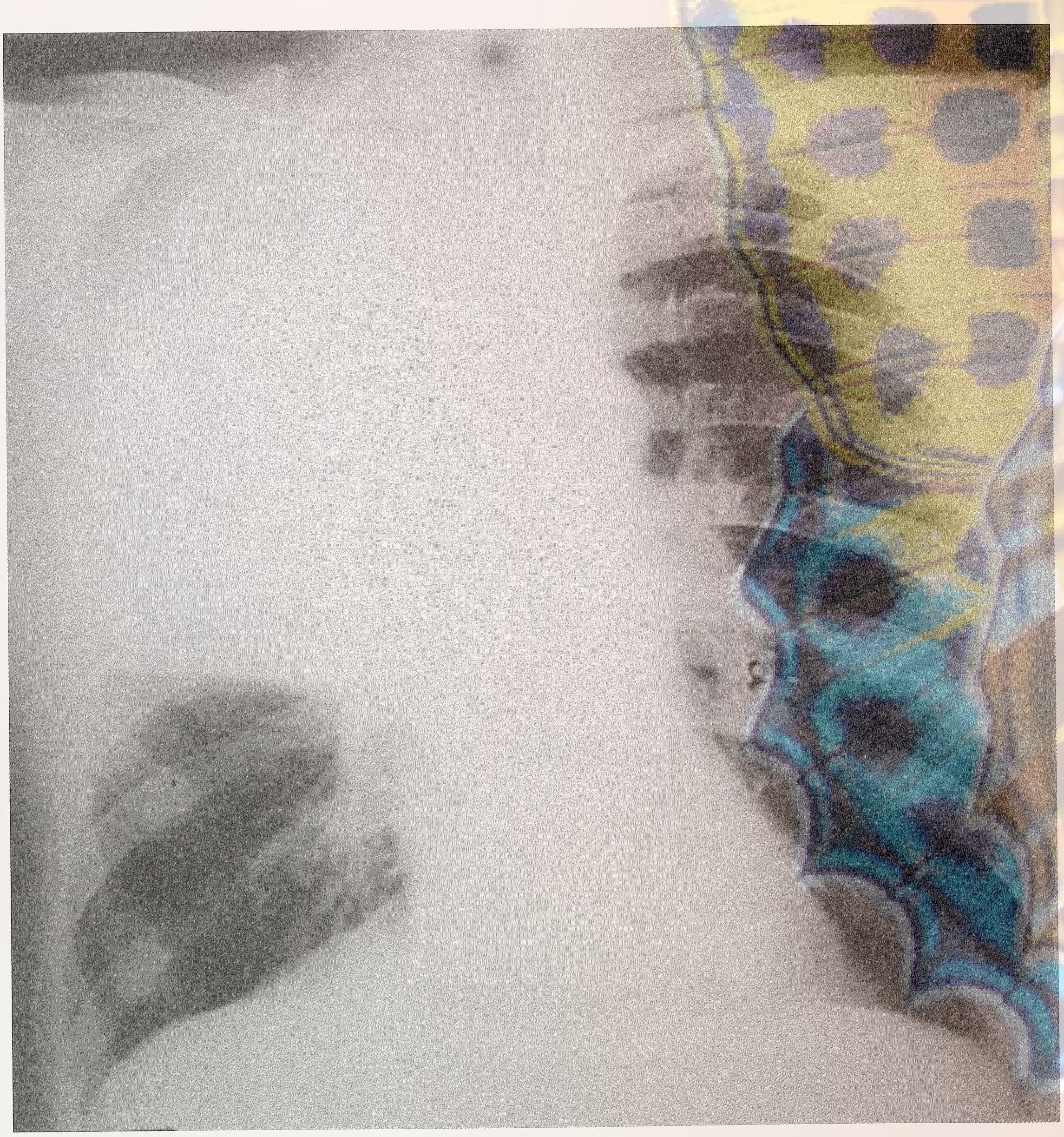
I. IMAGING: CXR

- Homogenous opacity: unilateral, usually involving one whole lobe.
- Complication: **e.g.** *pleural effusion*, *lung abscess*.

II. LABORATORY INVESTIGATIONS:

1. **Sputum examination (C&S):**
   - Pneumococci appear as: *Gm + ve diplococci*.
2. **Blood picture:**
   - Leukocytosis: *with shift to the left*.
3. **Blood culture:**
   - Pneumococci: *may be detected*.
4. **ABG:**
   - May show: *Hypoxia & Hypocapnia*.
5. **INVESTIGATIONS FOR COMPLICATIONS:**
   - Culture of aspirated pleural fluid.

Lobar pneumonia



DIFFERENTIAL DIAGNOSIS

- Causes of: ***acute high fever*** with chest symptoms, **e.g.** acute lung abscess.
- Consolidation should be differentiated from:
  Collapse, ***fibrosis***, ***pleural effusion***.
- Differentiate between: *lobar pneumonia* & *bronchopneumonia*.
- FUO.

TREATMENT

1. General Treatment:

- Bed rest.
- Proper nutrition.

2. Specific Treatment: *(antibiotics)*

- Crystalline penicillin G: 1 million u / 6h IV (7 days).
- In allergy to penicillin:
  - *Erythromycin:* 500 mg / 6 h IV (7 days) **or:**
  - *Cephalosporins*.
- In resistant cases: antibiotics are given according to C & S.

3. Symptomatic Treatment:

- Analgesics & Antipyretics.
- For early dry cough: cough sedatives, **e.g.** codeine phosphate.
- For productive cough:
  - *Mucolytics:* **e.g.** Bromhexine.
  - *Expectorants:* **e.g.** Potassium iodide.
- Oxygen inhalation: in severe cases.

4. TREATMENT OF COMPLICATIONS:

- **e.g.** ttt of pleural effusion.

BRONCHOPNEUMONIA

CAUSATIVE ORGANISM

- BACTERIA:
    - Gm + ve cocci: *Staph. aureus.* *St. pyogenes.*
    - Gm - ve bacilli: *H. influenzae, Klebsiella, Pseudomonas, Legionella.*
    - Anaerobes.
    - Mycobacterium TB.
- Viruses:
    - Influenza virus, adenovirus, RSV, CMV.
- Mycoplasma.
- Chlamydia.
- Rickettsia.

- Fungi: **e.g.** *Histoplasma, Candida.*
- Protozoa: **e.g.** *Pneumocystis carinii.*
- Parasites: **e.g.** *Filaria, Toxoplasma, Ascaris.*

PREDISPOSING FACTORS

- AGE: *extremes of age* (children & elderly).
- UNDERLYING CHEST DISEASE: *e.g. chronic bronchitis.*
- ENVIRONMENT: *cold weather, overcrowding.*
- ↓↓ IMMUNITY: *malnutrition, hyposplenism.*
- HOSPITALIZED PATIENTS.

PATHOLOGY

A) LOCATION:

- Diffuse bilateral patchy inflammation of: ***multiple lobules.***
  (affecting mainly: the alveoli adjacent to the bronchioles).

B) LESION:

- Lobular consolidation & may be bronchiolitis & bronchitis.

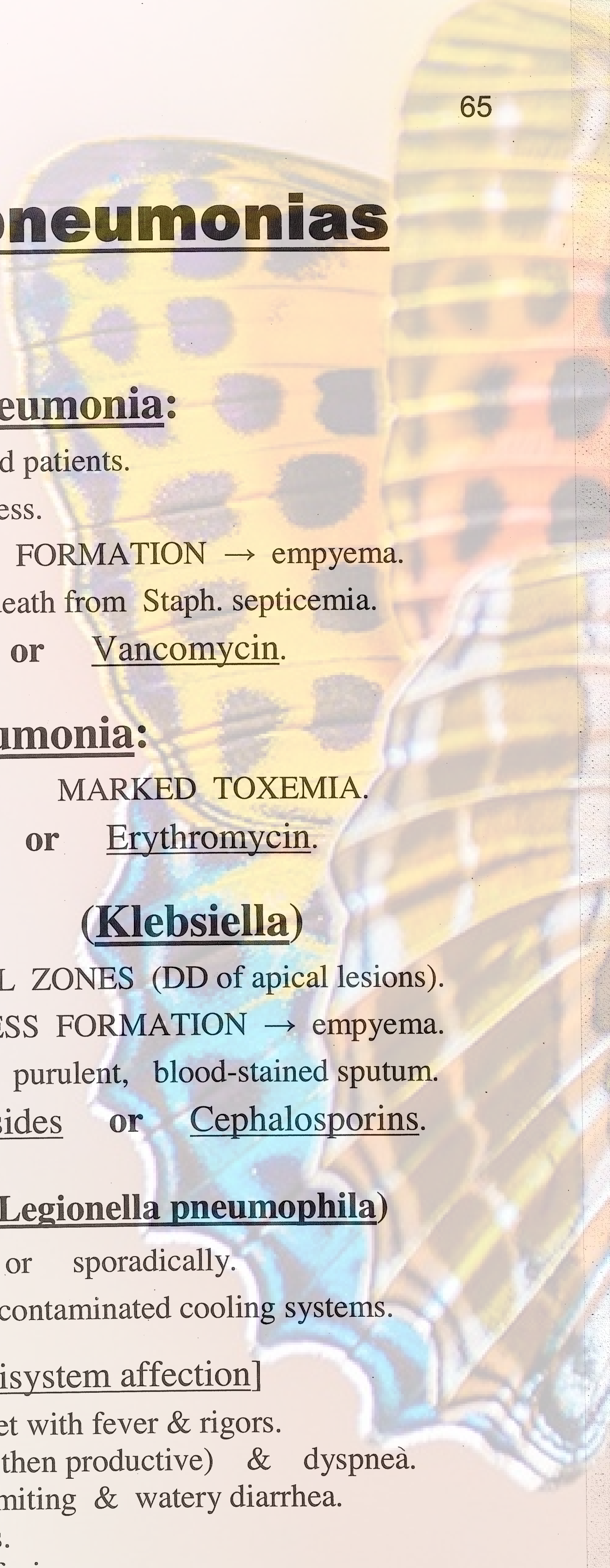
DIAGNOSIS

Similar to lobar pneumonia with the following differences:

	Lobar Pneumonia	Bronchopneumonia
Age	Middle	Extremes
Onset	Acute	More gradual
Offset	By crisis	By lysis
Duration	1 week	2 weeks
Pathology	Lobar consolidation	Lobular consolidation & bronchitis
Complications	Less common	More common
CXR	Lobar consolidation	Bilateral patchy opacities

TREATMENT

- Same lines of treatment of Lobar pneumonia, **BUT**:
- Specific ttt (Antibiotics): depends on the causative organism (see later).



Special types of pneumonias

1. BACTERIAL

a) Staphylococcal bronchopneumonia:

- Commonly occurs in: hospitalized patients.
- Commonly follows: a viral illness.
- There is tendency to: ABSCESS FORMATION → empyema.
- May take a fatal course leading to death from Staph. septicemia.
- ***Treatment***: Cloxacillin **or** Vancomycin.

b) Streptococcal bronchopneumonia:

- Characteristically associated with: MARKED TOXEMIA.
- ***Treatment***: Penicillin G **or** Erythromycin.

c) Friedlander's pneumonia: (Klebsiella)

- Characteristically affects: APICAL ZONES (DD of apical lesions).
- There is tendency to: ABSCESS FORMATION → empyema.
- Characteristically there is: viscid, purulent, blood-stained sputum.
- ***Treatment***: Aminoglycosides **or** Cephalosporins.

d) Legionnaire's disease: (Legionella pneumophila)

- Occurs in: outbreaks or sporadically.
- Spreads usually in: places with contaminated cooling systems.
- ***Clinical picture:*** [Multisystem affection]
    - *General:* gradual onset with fever & rigors.
    - *Chest:* cough (dry then productive) & dyspnea.
    - *GIT:* nausea, vomiting & watery diarrhea.
    - *Heart:* myocarditis.
    - *Neurological:* mental confusion.
    - *Renal:* ARF.
- ***Investigations***:
    - *CXR:* Lobar consolidation **or** Bilateral patchy opacities.
    - *Sputum:* organism is detected with special stain: **Gimenez**.
    - *Serology:* Immunofluorescent antibody (IFA): **↑ Ab titre**.
- ***Treatment***: **C**larithromycin **or** **C**iprofloxacin.

2. NON - BACTERIAL

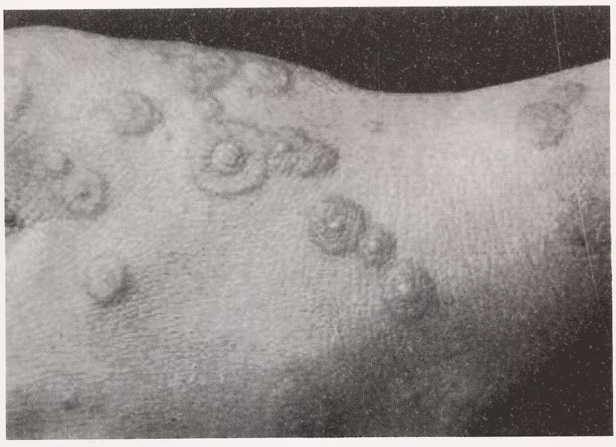
a) Viral pneumonia:

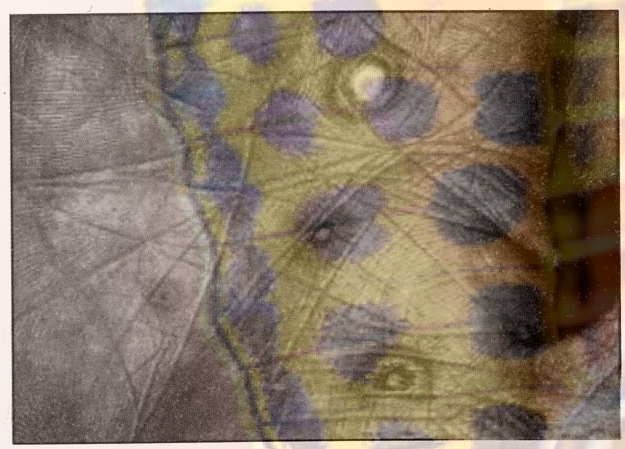
- The most common cause of pneumonia in: children.
- Causative organisms: *Influenza virus, adenovirus, varicella, RSV, CMV.*
- ***Clinical picture:***
  - *General:* Flu – like symptoms & may be skin rash.
  - *Chest:* dry cough, dyspnea, few physical signs.
- ***Investigations:***
  - *CXR:* may show bilateral patchy opacities.
  - *Serology:* antibodies to specific viruses.
- ***Treatment:*** Antiviral drugs: e.g. **R**ibavirin for **R**SV.

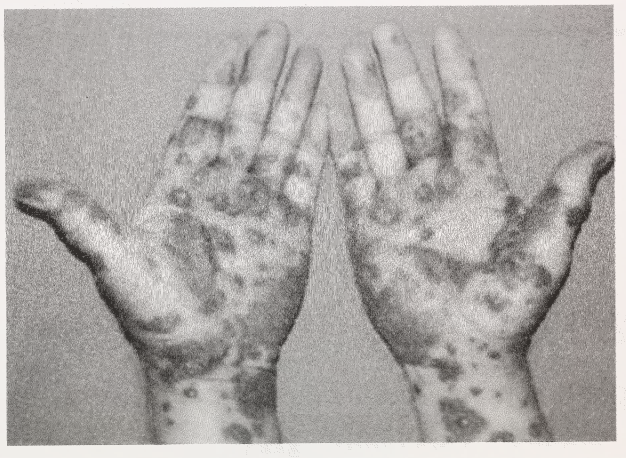
b) Atypical pneumonia:

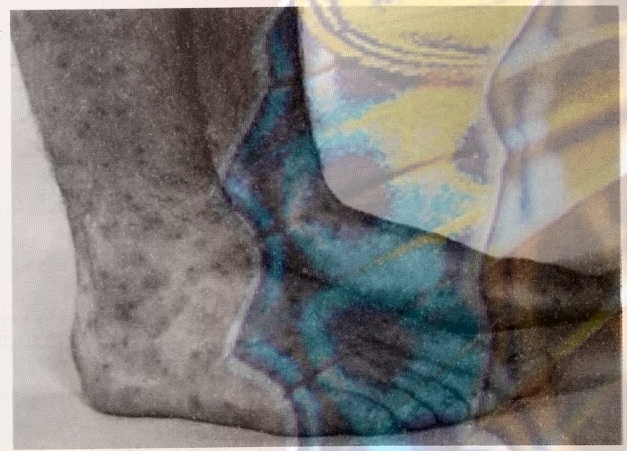
- Causative organisms: *MYCOPLASMA, chlamydia, rickettsiae.*
- ***Clinical picture:***
  - *Onset:* **more gradual onset with a rather mild fever**
  - *Chest:*
    1. Cough (dry, may be productive) & dyspnea.
    2. Minimal physical signs on the chest (*few crepitations*).
  - *Extrapulmonary:*
    1. General: fatigue, headache, myalgias.
    2. Skin: erythema multiforme.
    3. Blood: AIHA (*cold agglutinins*).
    4. Neurological: meningitis & encephalitis.
- ***Investigations:***
  - *CXR:* Severe abnormalities on CXR despite minimal physical chest signs: " Bilateral patchy opacities or GGA".
- ***Treatment:*** **C**larithromycin **or** **T**etracyclin.

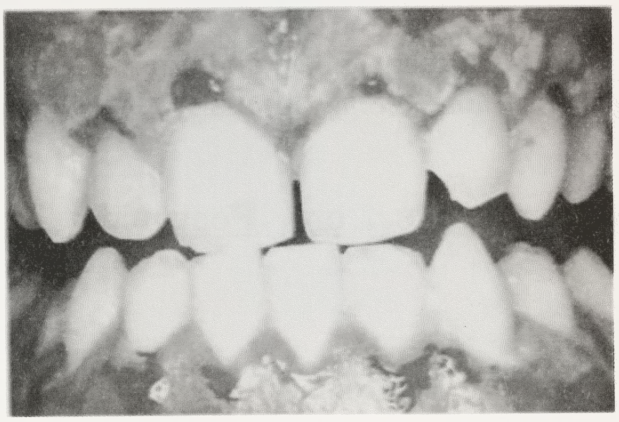
Erythema Multiforme

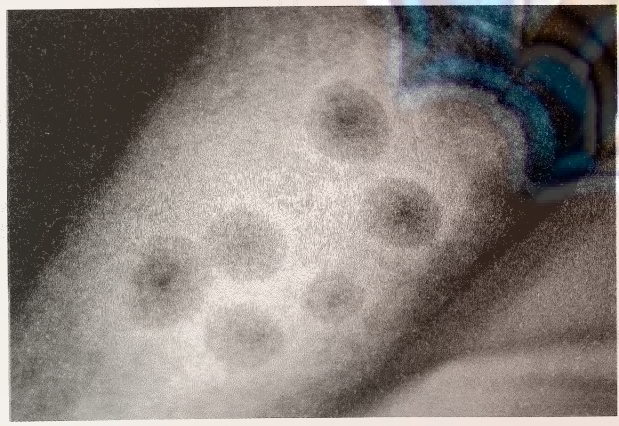


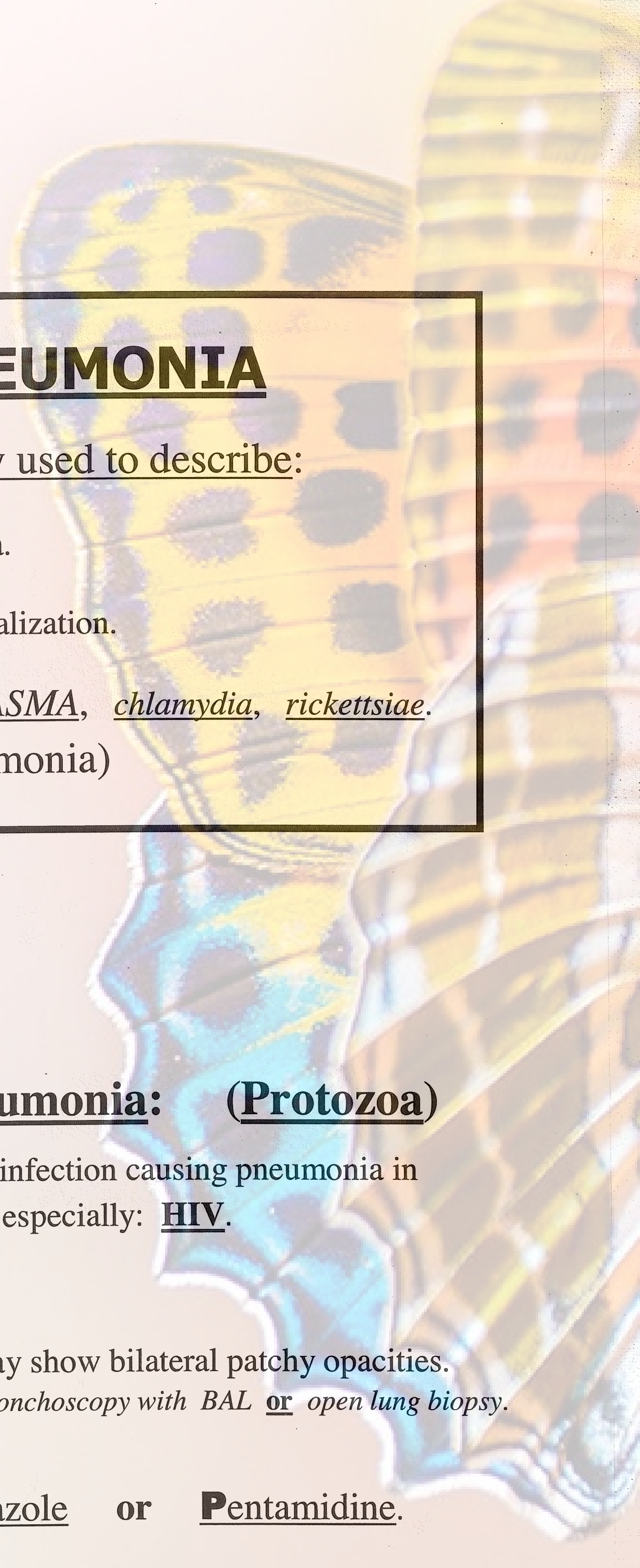












WALKING PNEUMONIA

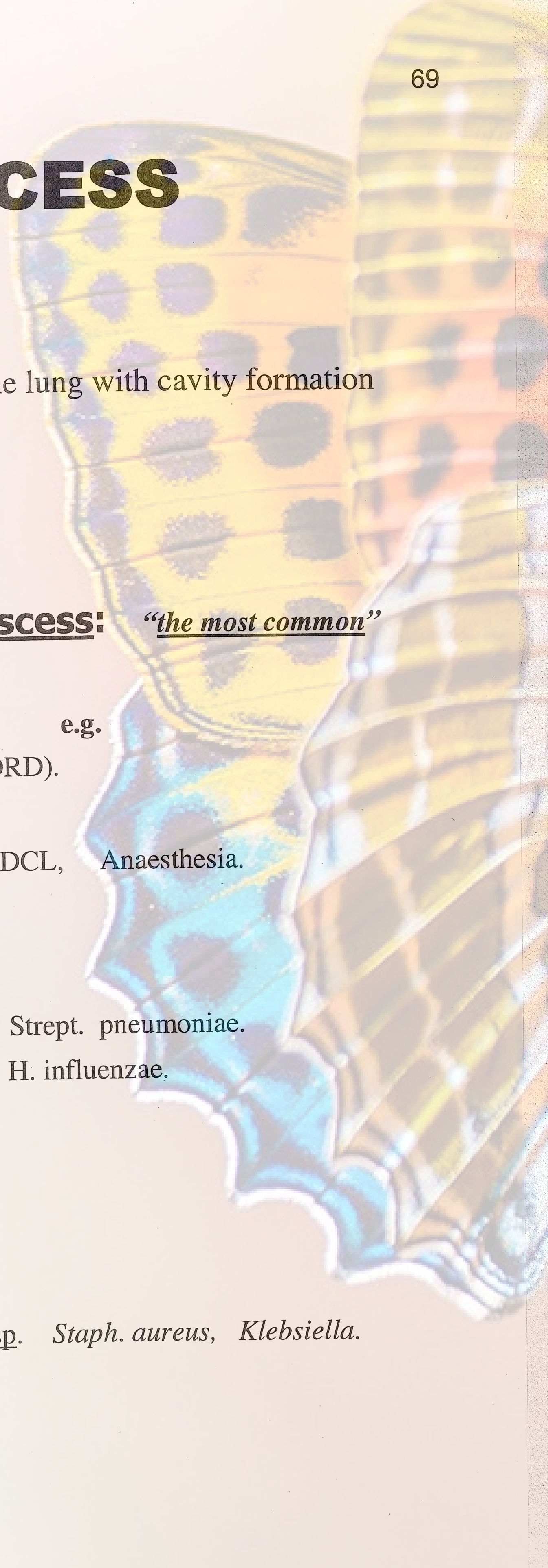
- An old term that was previously used to describe:
  - A clinically mild form of pneumonia.
  - Affected patients do not need hospitalization.
  - Causative organisms: *MYCOPLASMA*, *chlamydia*, *rickettsiae*.

(Atypical pneumonia)

c) Pneumocystis carinii pneumonia: (Protozoa)

- The most common opportunistic infection causing pneumonia in immunocompromised patients, especially: **HIV**.
- ***Diagnosis:***
  - CXR: may show bilateral patchy opacities.
  - Detection of organism: *Bronchoscopy with BAL* **or** *open lung biopsy.*
- ***Treatment*:** **C**o-trimoxazole **or** **P**entamidine.

LUNG ABSCESS



DEFINITION

- It is a localized **SUPPURATION** in the lung with cavity formation and fluid level, ***that is not due to TB***.

ETIOLOGY

1. Primary (inhalation) lung abscess: ***"the most common"***

- **Risk factors:**
  - a) Inhalation of a specific material: **e.g.**
    - FB, vomitus (e.g. GORD).
  - b) Absent cough reflex:
    - Stroke, Bulbar palsies, DCL, Anaesthesia.

- **Causative organisms:**
  - Anaerobes.
  - Gm +ve: Staph. aureus, Strept. pneumoniae.
  - Gm – ve: Klebsiella, H. influenzae.

2. Secondary lung abscess:

A. Thoracic causes:

1] Lung disease:

- Pneumonia, esp. *Staph. aureus, Klebsiella.*
- Bronchiectasis.
- Bronchial carcinoma.
- Lung collapse.

2] Mediastinal disease:

- Cancer oesophagus: infiltrating the lung.

3] **Subdiaphragmatic disease:**

- Amoebic liver abscess.
- Subphrenic abscess.

4] **Chest wall disease:**

- Penetrating wounds.
- Osteomyelitis of: *sternum, ribs or vertebrae.*

B. Extra – thoracic causes:

- **Pyaemia:** (***pyaemic abscess***)
  - May occur due to infective endocarditis, osteomyelitis.
  - The causative organism is usually: *Staph. aureus.*

PATHOLOGY

1. Primary (inhalation) lung abscess:

A. Site:

- Solitary.
- More common in the ***right lower lobe*** because the right bronchus is wider, and in direct continuity with the trachea.

B. Stage:

1) **Pneumonic stage:**
   - There is an area of ***consolidation*** (pneumonic patch), and if it is superficial, the covering pleura will show ***acute pleurisy***.

2) **Stage of acute abscess:**
   - Tissue necrosis occurs in the consolidated area producing a ***cavity*** with a necrotic wall.
   - The covering pleura shows ***acute pleurisy***.

3) **Stage of chronic abscess:**
   - The wall of the ***cavity*** becomes thickened by ***fibrosis***.
   - The covering pleura may show fibrous adhesions.

2. Pyaemic abscesses:

- They are usually: *bilateral, multiple, small & of equal sizes.*

CLINICAL PICTURE

1. Pneumonic stage: *same as in lobar pneumonia*

2. Stage of acute abscess:

Symptoms

- After 1 – 2 weeks, the patient suffers from an attack of severe cough followed by expectoration of: ***excessive purulent foeted sputum***.
- The general condition improves & the temperature drops.
- The case continues as a: SUPPURATIVE SYNDROME:
  Paroxysmal cough with expectoration of: ***excessive purulent foeted sputum*** related to posture when the patient lies on the ***healthy side***.
- Haemoptysis may occur.

Signs

- **General signs:** Fever, tachycardia & sweating.
- **Chest signs:** Features of a ***cavity*** & may be ***pleurisy***.

Inspection

- Shape: normal shape of the chest.
- Movements: limited on the affected side.

Palpation

- Trachea & mediastinum: central.
- TVF: increased.

Percussion

- Dullness: on the affected area, due to ***cavitation***.
- Tenderness: on the affected area, due to ***pleurisy***.

Auscultation

- Breath sound: bronchial breathing.
- Additional sounds: crepitations: **Medium sized** **Coarse**
- Pleural rub: is usually present.

3. Stage of chronic abscess: (Duration > 2 months)

Symptoms

- Symptoms of: SUPPURATIVE SYNDROME.
- Attacks of: RETENTION SYNDROME:
  - The draining bronchus becomes obstructed with thick secretions.
  - So fever rises and sputum diminishes.
  - After few days the patient coughs repeatedly and expectorates the obstructing plug and symptoms of suppurative syndrome reappear.
- Progressive DETERIORATION OF GENERAL HEALTH.

Signs

General signs:

- Recurrent fever.
- Pallor, toxic face, loss of weight.
- Puffiness of eye lids: *due to chronic cough*.
- Clubbing of fingers: *due to chronic toxemia*.

Chest signs: ***(Signs of cavity & may be fibrosis)***

Inspection

- Shape: Normal, but may be retraction due to ++ fibrosis.
- Movements: Limitation of movements on the affected side.

Palpation

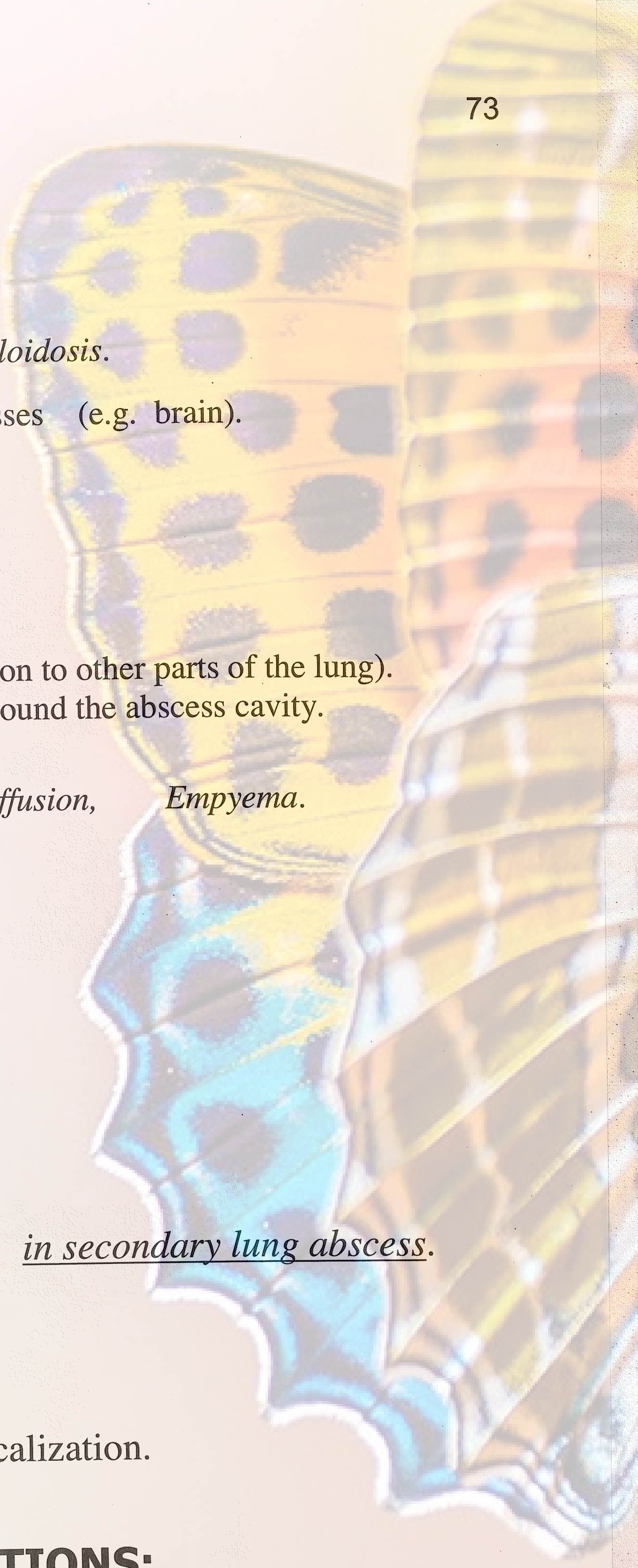
- Trachea & mediastinum: Central, but may be shifted to the same side due to ++ fibrosis.
- TVF: Increased.

Percussion

- Dullness: on the affected area, due to ***cavitation***.

Ausculation

- Breath sound: bronchial breathing.
- Additional sounds: crepitations: **Medium sized** **Coarse**



COMPLICATIONS

A. General complications

- *Toxemia:* may lead to *amyloidosis.*
- *Bacteremia:* metastatic abscesses (e.g. brain).
- *Anemia.*

B. Chest complications

- *Lung:*
    - Pneumonia (spread of infection to other parts of the lung).
    - Fibrosis & bronchiectasis around the abscess cavity.
- *PLEURA:* *Pleurisy,* *Pleural effusion,* *Empyema.*

INVESTIGATIONS

I. IMAGING:

1) CXR:

- Cavity with a fluid level.
- May show the cause: *in secondary lung abscess.*
- May show complications.

2) CT chest:

- For accurate diagnosis & localization.

II. LABORATORY INVESTIGATIONS:

1) Sputum examination (C&S):

- To detect the causative organism.

2) Blood picture:

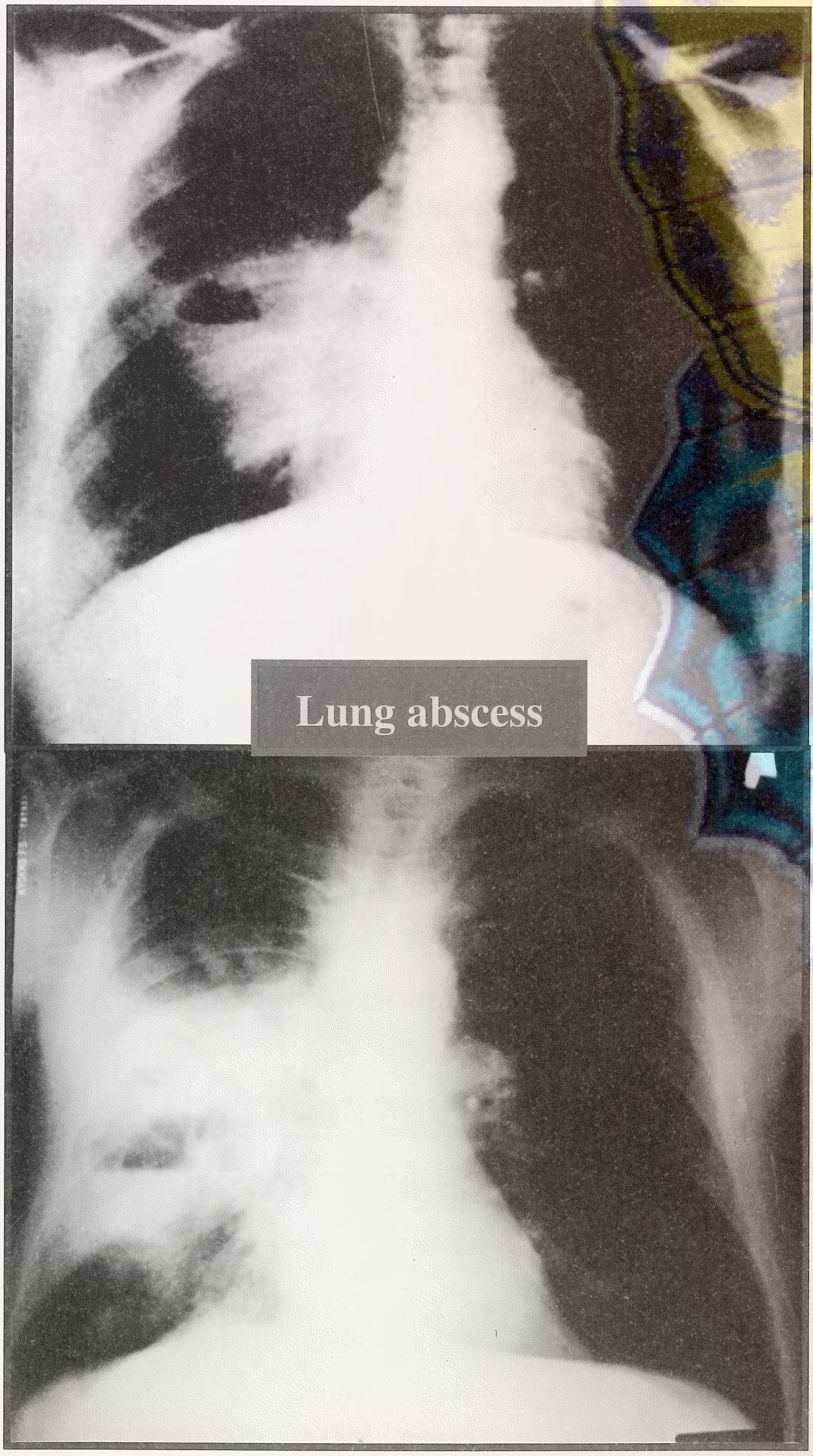
- Leukocytosis: *with shift to the left.*

3) INVESTIGATIONS FOR COMPLICATIONS:

- Culture of aspirated pleural fluid.

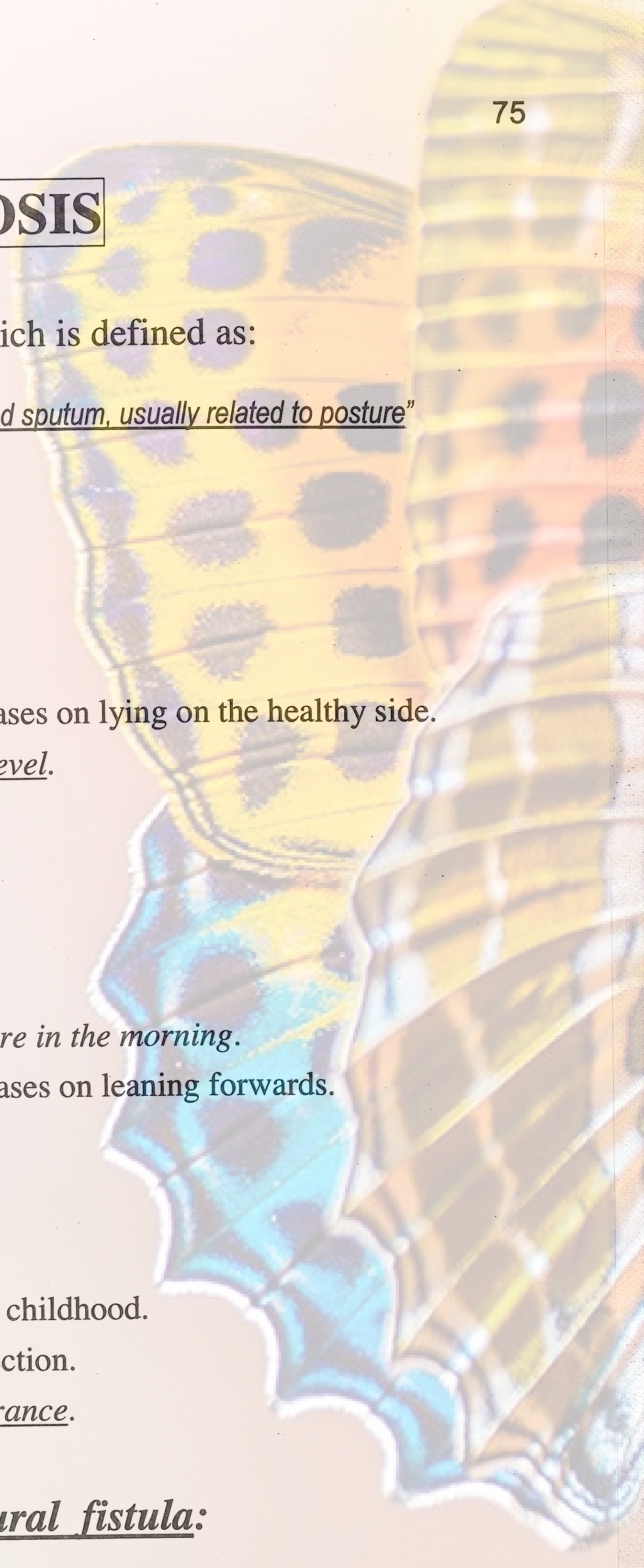
III. BRONCHOSCOPY: *indicated to:*

- Localize the site of the abscess
- Exclude the presence of underlying malignancy.
- Detect & remove foreign body.
- Drain pus & inject antibiotics into the cavity.



Lung abscess

DIFFERENTIAL DIAGNOSIS



A. Causes of SLS: which is defined as:

"Cough with expectoration of excessive purulent foeted sputum, usually related to posture"

1. *Lung abscess:*

- Site: localized.
- Onset: acute.
- Periodicity: *absent.*
- Posture: expectoration increases on lying on the healthy side.
- CXR: *cavity with a fluid level.*

2. *Bronchiectasis:*

- Site: bilateral & basal.
- Onset: gradual.
- Periodicity: *more in winter , more in the morning.*
- Posture: expectoration increases on leaning forwards.
- CXR: *honey-combing.*

3. *Infected cystic lung:*

- Signs of SLS since birth or in early childhood.
- Associated liver & pancreatic affection.
- CXR: *soap-bubble appearance.*

4. *Empyema with broncho – pleural fistula:*

- Signs of pyopneumothorax, with:
  - Shifting dullness
  - Positive Methylene blue test.

B. Tuberculous cavity.

TREATMENT

1. Drainage of pus:

1) Physical therapy & Postural drainage.
2) Bronchoscopic aspiration & injection of antibiotics may be used.

2. Control of infection:

- Antibiotics according to sputum C & S.
- The most commonly used antibiotics are:

For anaerobes:	Clindamycin or Metronidazole.
For Gm +ve, Staph. aureus:	Cloxacillin or Vancomycin.
For Gm +ve, St. pneumoniae:	Penicillin G or Erythromycin.
For Gm – ve, Klebsiella:	Aminoglycosides or Cephalosporins.
For Gm – ve H. influenzae:	Ampicillin.

3. Symptomatic Treatment:

- Analgesics & Antipyretics.
- For productive cough:
  - *Mucolytics:* **e.g.** Bromhexine.
  - *Expectorants:* **e.g.** Potassium iodide.

4. TREATMENT OF COMPLICATIONS:

- **e.g.** ttt of pleural effusion.

5. Surgery (segmentectomy or lobectomy) in:

- Chronic or huge abscess not responding to medical treatment.
- Severe recurrent hemoptysis.
- Presence of underlying malignancy
- Complicating empyema or severe bronchiectasis.

BRONCHIECTASIS

DEFINITION

- Abnormal & permanent dilatation of the bronchi.

PATHOLOGY “2”

I. Lower parts:

- *SITE*:
  - Bilateral & Basal.
- *BRONCHI*:
  - Are: inflamed & dilated
  - Full of: mucus & pus.
- *SURROUNDING LUNG TISSUE*:
  - May show: consolidation & fibrosis.

II. Upper parts:

- Bilateral emphysema of the upper parts of the lungs occur due to chronic bronchitis resulting from aspiration of infected sputum into the healthy bronchi.

ETIOLOGY “2”

1. Congenital bronchiectasis:

a) Isolated.

b) Part of a syndrome:

1) ***Immotile cilia syndrome***:
   - Bronchiectasis.
   - Sinusitis.
   - Male sterility.

2) ***Kartagner’s syndrome***:
   - Bronchiectasis.
   - Sinusitis.
   - Dextrocardia.

2. Acquired bronchiectasis:

A. Infection & Fibrosis:

- Recurrent infections lead to destruction & weakening of the musculo-elastic tissue in the wall of the bronchi resulting in their dilatation.

- In addition, peribronchial fibrosis pull on the bronchi from outside leading to further dilatation.

- The **most common** infections causing bronchiectasis are:
  - Viruses : *Adenovirus.*
  - Bacteria: *Staph.aureus, Klebsiella, Anaerobes,* ***H. influenzae, Strept. pneumonae.***
  - Mycobacteria: *TB.*

B. Bronchial obstruction: which may be either,

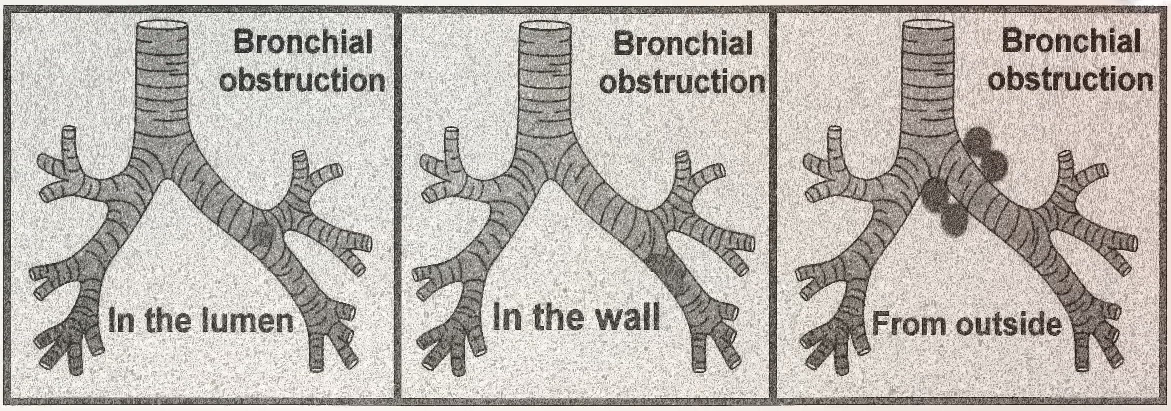
a) Partial:

- It acts as a valve mechanism allowing air to enter during inspiration, but preventing it to escape during expiration, so the intra-bronchial pressure will rise progressively leading to bronchiectasis.

b) Complete:

- Collapse resulting from bronchial obstruction increases the negativity of the intrapleural pressure which exerts traction on the bronchi resulting in their dilatation.

- **Causes of bronchial obstruction:**
  - In the lumen: *FB or thick secretions.*
  - In the wall: *Tumour.*
  - Pressure from outside: *Tumour, LN.*



CLINICAL PICTURE

Symptoms

A. Cough & expectoration: *(Suppurative lung syndrome)*

- Persistent recurrent cough with expectoration of suptum which is:
  - Excessive.
  - Mucorpurulent.
  - Foetid.
  - Increases in the morning & on leaning forwards.
- Onset is gradual & the course is progressive over years with characteristic winter exacerbations.

B. Hemoptysis:

- It may be blood-tinged or frank.
- It may be the only presenting symptom ***"bronchiectasis sicca hemorrhagica"***, esp. in cases secondary to **TB**.

C. Chest Pain: *due to:*

- Muscular pain because of severe cough.
- Pleuritic pain with occurrence of pleurisy.

D. Dyspnea: *due to:*

- Fibrosis.
- Obstruction.

E. Wheezing:

- Reflects extensive bronchiectasis.

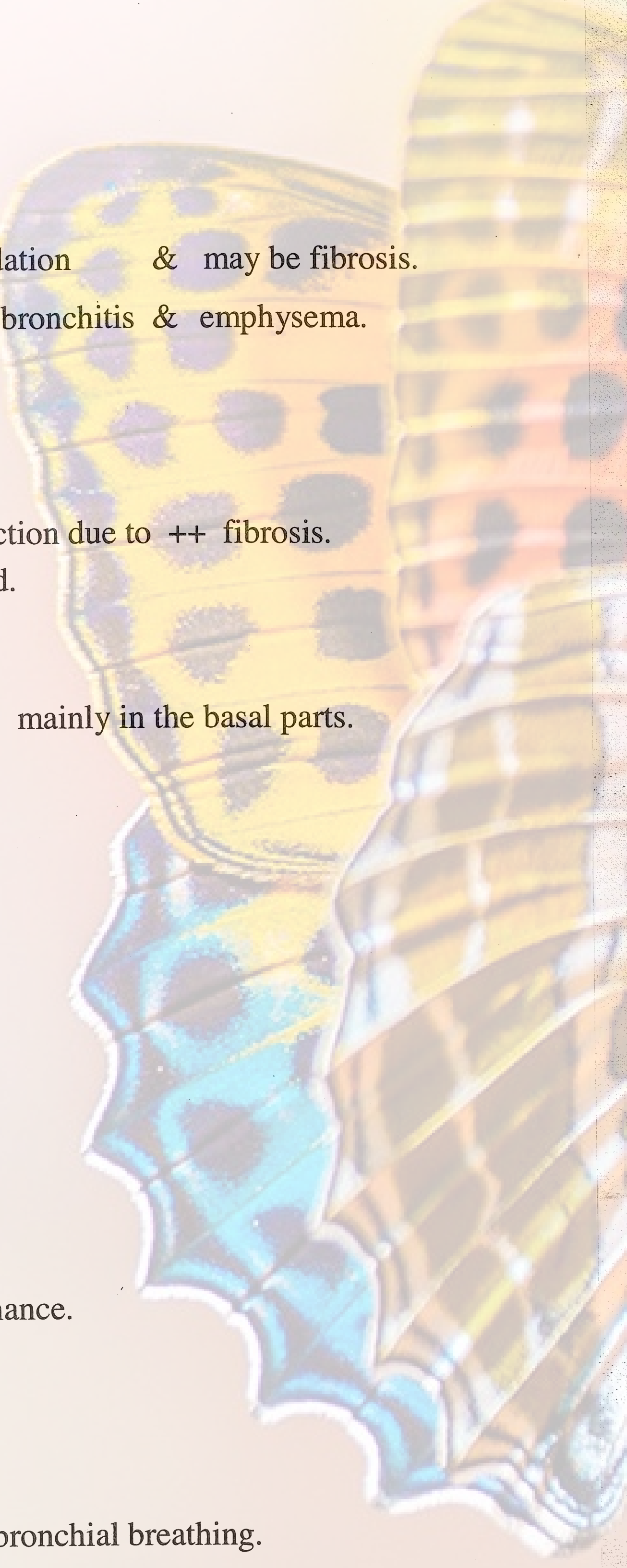
Signs

1. General signs:

- Recurrent fever.
- Pallor, toxic face, loss of weight.
- Puffiness of eye lids: *due to chronic cough*.
- Clubbing of fingers: *due to chronic toxemia*.
- OEDEMA OF LLs: *due to*,
  - Cor pulmonale & RVF.
  - Renal amyloidosis.
  - Excessive loss of proteins in the sputum.

2. Chest signs:

- In the **basal parts:** signs of consolidation & may be fibrosis.
- In the **upper parts:** signs of chronic bronchitis & emphysema.



Inspection

- Shape:

  Basal parts: may be retraction due to ++ fibrosis.
  Upper parts: Hyperinflated.

- Movements:

  Limitation of movements, mainly in the basal parts.

Palpation

- Trachea & mediastinum:

  Central.

- TVF:

  Basal parts: ↑.
  Upper parts: ↓.

Percussion

- Basal parts: Patchy dullness.
- Upper parts: May be hyper-resonance.

Ausculation

- Basal parts:
  - Breath sound: areas of bronchial breathing.
  - Additional sounds: crepitations: **Medium sized** **Coarse**
- Upper parts:
  - Breath sound: Vesicular breathing with prolonged expiration.
  - Additional sounds: Rhonchi.

COMPLICATIONS

A. General complications

- *Toxemia:* may lead to *amyloidosis.*
- *Bacteremia:* metastatic abscesses (e.g. brain).
- *Anemia.*

B. Chest complications

- *Lung:*
  - Pneumonia (spread of infection to other parts of the lung).
  - Lung abscess (spread of infection to other parts of the lung).
- *PLEURA:* *Pleurisy,* *Pleural effusion,* *Empyema.*
- *Pulmonary hypertension:* → corpumonale & RVF.
- *Respiratory failure:* due to associated emphysema.

INVESTIGATIONS

I. IMAGING:

1) CXR:

1. Basal parts:
   - Prominent cystic spaces having the characteristic: " *Honey-comb appearance* ".
   - Opacities around the cysts due to consolidation.
2. Upper parts: " *Hypertranslucency* " due to emphysema.

2) CT chest: *(the most important investigation)*

- It provides excellent visualization of the bronchiectatic airways.
- It localizes the site & extent of bronchiectasis before surgery.

3) Bronchography:

- It provides excellent visualization of the bronchiectatic airways.
- It shows the dilated bronchi as: *cylindrical, saccular or fusiform.*

II. LABORATORY INVESTIGATIONS:

1) Sputum examination (C&S):

- To detect the causative organism.

2) Blood picture:

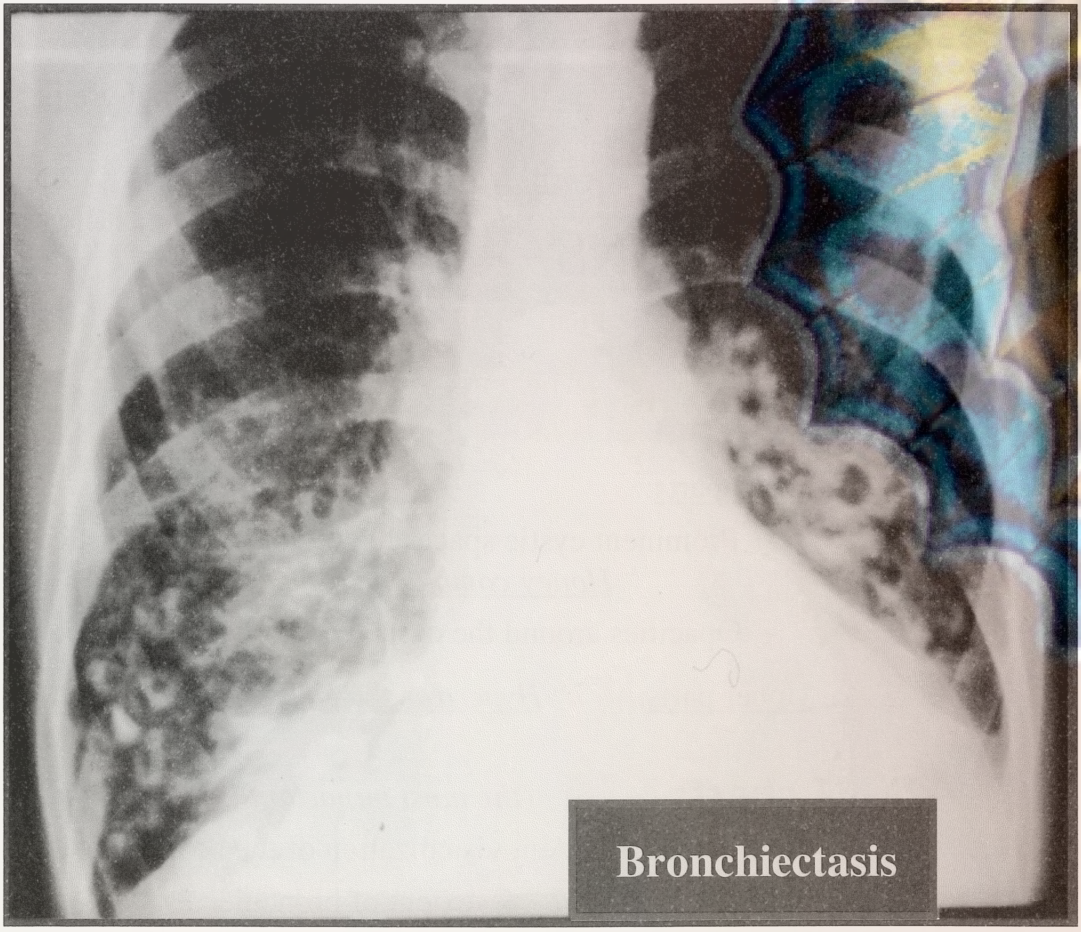
- Leukocytosis: *with shift to the left*.

3) INVESTIGATIONS FOR COMPLICATIONS:

- Culture of aspirated pleural fluid.

III. BRONCHOSCOPY: *indicated to*:

- May reveal the cause in localized bronchiectasis.
- May be used for drainage of pus & injection of antibiotics.



Bilateral & basal:

- **Prominent cystic spaces (Honey – comb appearance)**
- **Opacities around the cysts due to consolidation**

DIFFERENTIAL DIAGNOSIS

- Causes of suppurative lung syndrome.
- Causes of hemoptysis.

TREATMENT

1. **Drainage of pus:**

1) Physical therapy & Postural drainage.
2) Bronchoscopic aspiration & injection of antibiotics may be used.

2. **Control of infection:**

- Antibiotics according to sputum C & S.
- The most commonly used antibiotics are:

☒ For anaerobes:	Clindamycin or Metronidazole.
☒ For Gm +ve, Staph. Aureus:	Cloxacillin or Vancomycin.
☒ For Gm – ve, Klebsiella:	Aminoglycosides or Cephalosporins.
☒ For H. influenzae:	Ampicillin.
☒ For Strept. pneumonae:	Crystalline penicillin G.

3. **Symptomatic Treatment:**

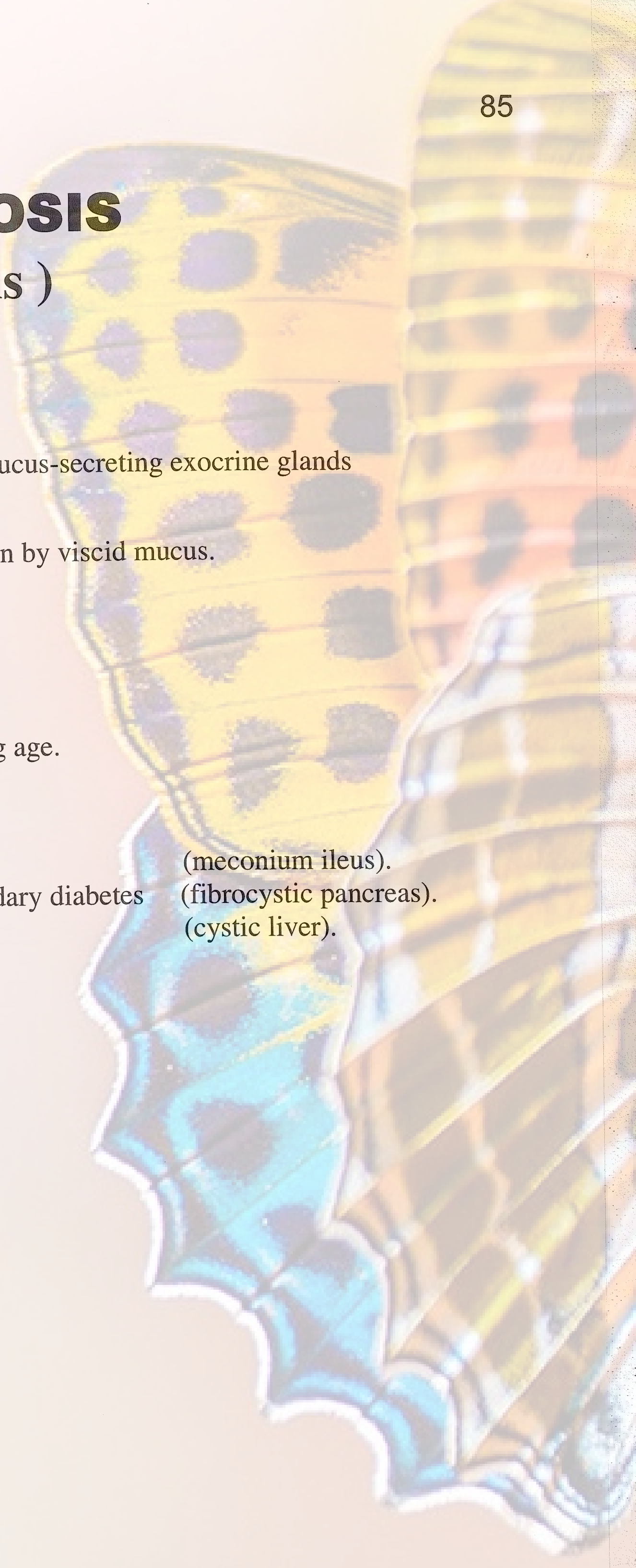
- Analgesics & Antipyretics.
- For productive cough:
  - *Mucolytics:* e.g. Bromhexine.
  - *Expectorants:* e.g. Potassium iodide.
- For airway obstruction:
  - *Bronchodilators.*

4. **TREATMENT OF COMPLICATIONS:**

- e.g. ttt of pleural effusion.

5. Surgery (local resection or lobectomy) in:

- Persistent or recurrent infection not responding to medical treatment.
- Severe recurrent hemoptysis.
- Localised bronchiectasis.



CYSTIC FIBROSIS
(Mucoviscidosis)

DEFINITION

- A hereditary disease (autosomal recessive), affecting mucus-secreting exocrine glands and sweat glands.
- Most of the clinical features are secondary to obstruction by viscid mucus.

CLINICAL PICTURE

1) **Chest (Polycystic lung):**
   - Recurrent infections & SLS, usually in a young age.

2) **GIT & Liver:**
   - Intestinal obstruction in the newly born (meconium ileus).
   - Pancreatic insufficiency & malabsorption, secondary diabetes (fibrocystic pancreas).
   - Jaundice & later cirrhosis (cystic liver).

3) **Genitourinary:**
   - Male sterility (obliteration of the vas deferens).

INVESTIGATIONS

1) **CXR:**
   - Soap-bubble appearance.
   - Multiple fluid levels in cases of infection.

2) **Pancreatic function tests:**
   - May be impaired.

3) **Sodium in Sweat:**
   - Is markedely elevated.

TREATMENT

1- *For polycystic lung*: same as bronchiectasis.

2- *For meconium ileus*:
   - Enemas & nasogastric suction.
   - Surgery is rarely needed.

3- *For pancreatic insufficiency*:
   - Pancreatic enzyme replacement.

RESPIRATORY FUNCTIONS

1. Ventilation.
2. Perfusion.
3. Diffusion.

VENTILATION

DEFINITION

- It is the process of: movement of air in & out of the lungs.
- It is the process of: exchange of air between the lungs and the atmosphere.
- It includes: **INSPIRATION** & **EXPIRATION**.

PHYSIOLOGY

INSPIRATION (breathing in): is an ACTIVE process.

- It occurs when the muscles of the diaphragm and chest wall contract.
- The contraction of these muscles will:
  1. Expand the ***chest wall***, the ***pleura***, the ***lung*** & therefore will allow air to enter.
  2. Lowers the pressure inside the chest, & thus lowers the pressure in the airways:
     - Pressure gradient occurs from mouth to alveoli, so air rushes in.

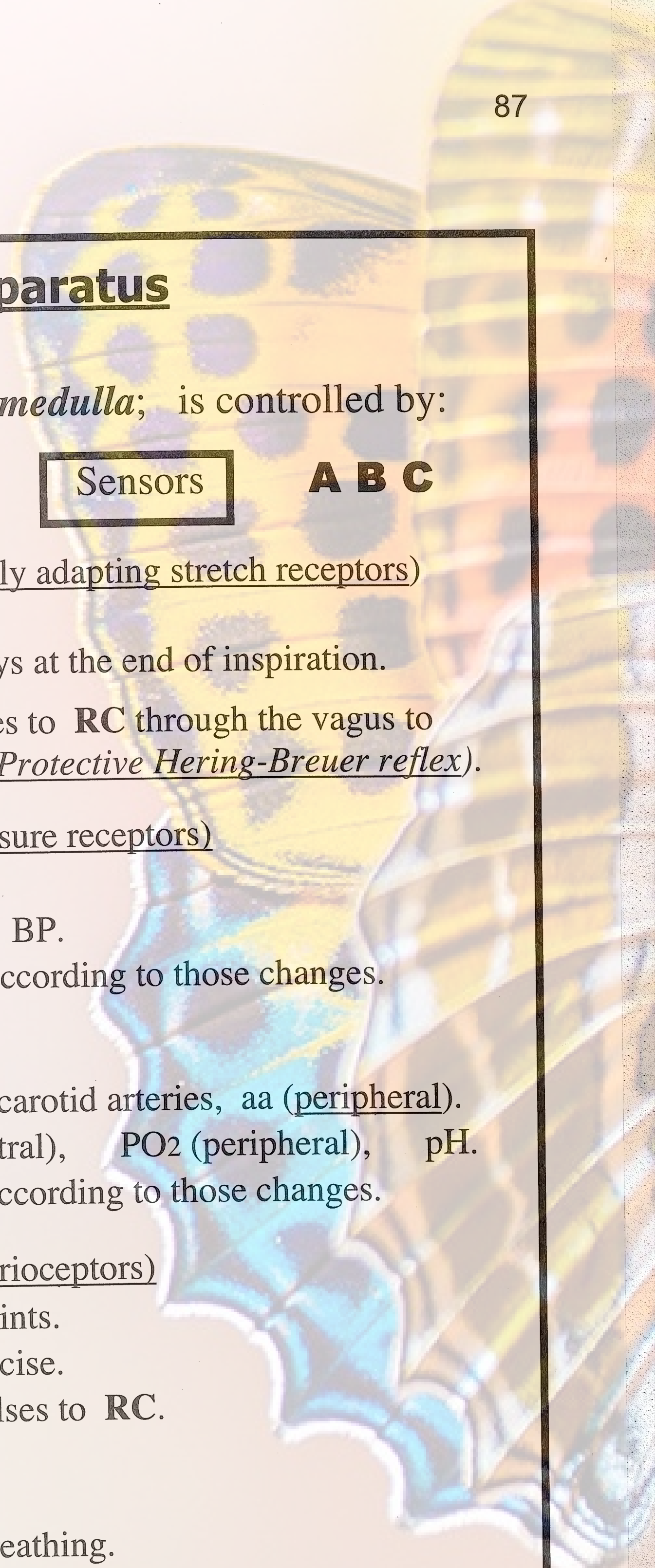
EXPIRATION (breathing out): is a PASSIVE process.

- As the muscles relax, the elastic recoil of the lungs puts pressure on the gases inside.
- The pressure in the chest is now higher than the outside pressure:
  - Pressure gradient occurs from alveoli to mouth, so air rushes out.

- **Ventilation will depend on:**

1. Diameter of the airways.

2. Compliance of: *chest wall*, *pleura*, *lungs*.
3. Neuromuscular apparatus.



Neuromuscular apparatus

I. RESPIRATORY CENTRE in the *medulla*; is controlled by:

A) Feedback from afferent receptors: Sensors **A B C**

1. **A**IRWAY RECEPTORS (Slowly adapting stretch receptors)
   - Location: walls of the bronchi.
   - Sensed by: expansion of the airways at the end of inspiration.
   - Function: send inhibitory impulses to **RC** through the vagus to terminate inspiration (*Protective Hering-Breuer reflex*).
2. **B**ARORECEPTORS (Pressure receptors)
   - Location: carotid sinus.
   - Sensed by: changes in the arterial BP.
   - Function: modulate ventilation according to those changes.
3. **C**HEMORECEPTORS
   - Location: medulla (central), carotid arteries, aa (peripheral).
   - Sensed by: changes in PCO_2 (central), PO_2 (peripheral), pH.
   - Function: modulate ventilation according to those changes.
4. OTHER RECEPTORS (Proprioceptors)
   - Location: muscles, tendons, joints.
   - Sensed by: movement during exercise.
   - Function: send stimulatory impulses to **RC**.

B) Cerebral cortex:

- Provides some voluntary control of breathing.

II. NEURAL PATHWAY: Conductors

• AHCs	(Cervical & Thoracic segments of the spinal cord).
• Respiratory nerves	(Phrenic & Intercostals nerves).
• NMJ.	

III. RESPIRATORY MUSCLES. Effectors

HYPOVENTILATION

ETIOLOGY

1. **Obstructive hypoventilation:** (↓ diameter of airways)

a. *Upper respiratory tract obstruction*: **e.g.**

- Inhaled FB, laryngeal spasm, laryngeal oedema.

b. *Lower respiratory tract obstruction*: **e.g.**

- Bronchial asthma.
- COPD.

2. **Restrictive hypoventilation:**

a. *Decreased compliance of*:

- Lungs: ILD.
- Pleura: Massive effusion , Tension pneumothorax.
- Chest wall: Chest deformities, scleroderma, marked obesity *(Pick synd)*.

b. *Disorders of neuromuscular apparatus*:

- CNS: Head injury, CVS, Drugs (*opiates & barbiturates*).
- AHCs: MND, Poliomyelitis.
- PN: Polyneuropathy, **e.g.** *GBS*.
- NMJ: Myasthenia gravis.
- Muscle: Myopathies.

MANIFESTATIONS

- Hypoxia.
- Hypercapnia.

DIFFUSION

DEFINITION

- It is the process of gas exchange between the alveolar air & the pulmonary capillary blood.
- It occurs through the alveolo – capillary membrane.

PHYSIOLOGY

- **Diffusion will depend on:**

1. **Ventilation – perfusion matching:**

   - ***Normally***, gas exchange depends on the proper matching of ventilation and perfusion, **i.e.** there should be equal distribution of the inspired air and the perfusing blood in different parts of the lungs (V / Q ratio).

   - ***Abnormally***, gas exchange will become impaired if there is V / Q mismatch.

Causes of Ventilation – perfusion mismatch

a. Increased V / Q ratio: (**dead space effect**)

- It means ventilation of unperfused areas of the lungs, e.g. *pulmonary embolism.*

b. Decreased V / Q ratio: (**shunt effect**)

- It means perfusion of unventilated areas of the lungs, e.g. *pneumonia.*

2. **Surface area of diffusion.**

3. **Intact alveolo-capillary membrane.**

4. **Pressures of O_2 in the alveolar air & CO_2 in the capillary blood.**

IMPAIRED DIFFUSION

ETIOLOGY

- ILD.
- Emphysema.
- Pulmonary oedema.

- Pulmonary embolism.
- Pneumonia.

MANIFESTATIONS

- Hypoxia.
- Normocapnia.

PERFUSION

- It is the process by which the pulmonary circulation delivers blood to the gas exchange units so that: O_2 uptake & CO_2 elimination can occur.

Respiratory Function Tests

I. Tests for specific pulmonary functions:

A. VENTILATION TESTS:

1. **Respiratory volumes & capacities:** (using the spirometer)
   - *See later.*
2. **Expiratory flow rates:**
   - *Forced expiratory volume in the first second* **(FEV_1)**:
     - Normally: ~ 3800 ml.
     - Normally: FEV_1 / FVC ~ 80 % (Timed vital capacity).
   - *Peak expiratory flow rate* **(PEFR)**.

These tests are impaired in: ***OBSTRUCTIVE HYPOVENTILATION***

3. **Lung compliance:**
   - *The ratio of change in volume of the lung to change in intrapleural pressure.*
4. **Ventilation scan:**
   - *Using radioactive Xenon* *(see pulmonary embolism).*

B. DIFFUSION TESTS:

- Diffusing capacity: (CO transfer factor)
  - It measures the quantity of CO that will diffuse in a time unit from the alveolar gas into the capillary blood.
  - It is a useful measure for the integrity of the alveolo – capillary membrane.

C. PERFUSION TESTS:

1. *Pulmonary vascular pressures.*
2. *Perfusion lung scan: using radioactive albumin (see pulmonary embolism).*

II. Tests for general pulmonary functions:

- ABG: *PO_2 & PCO_2.*
- Pulse Oximetry.

Tidal Volume: TV

- It is the volume of air inspired & expired during: ***normal quiet breathing***.
- Normally ~ 500 ml.
- Resting minute volume (RMV) = TV x RR (16 / min.) = 8000 ml / min.

Residual Volume: RV

- It is the volume of air ***remaining*** in the lung: ***after maximum expiration***.
- Normally ~ 1200 ml.
- ***It increases in:*** *COPD*.

Forced Vital capacity: FVC

- It is the volume of air that is ***maximally expired***: ***after maximum inspiration***.
- Normally ~ 4800 ml (ERV + IC)
- ***It decreases in:*** *all cases of hypoventilation, especially* ***restrictive hypoventilation.***

Total Lung Capacity: TLC

- It is the volume of air that the lungs contain when they are fully expanded.
- Normally ~ 6000 ml (RV + FVC)
- ***It decreases in:*** *restrictive hypoventilation.*

Expiratory Reserve Volume: ERV

- It is the additional volume of air that can be exhaled after a normal tidal breath out.
- Normally ~ 1200 ml.

Inspiratory Reserve Volume: IRV

- It is the additional volume of air that can be inhaled after a normal tidal breath in.
- Normally ~ 3100 ml.

Functional Residual Capacity: FRC

- It is the volume of air left in the lungs after a normal tidal breath out.
- Normally ~ 2400 ml (RV + ERV)

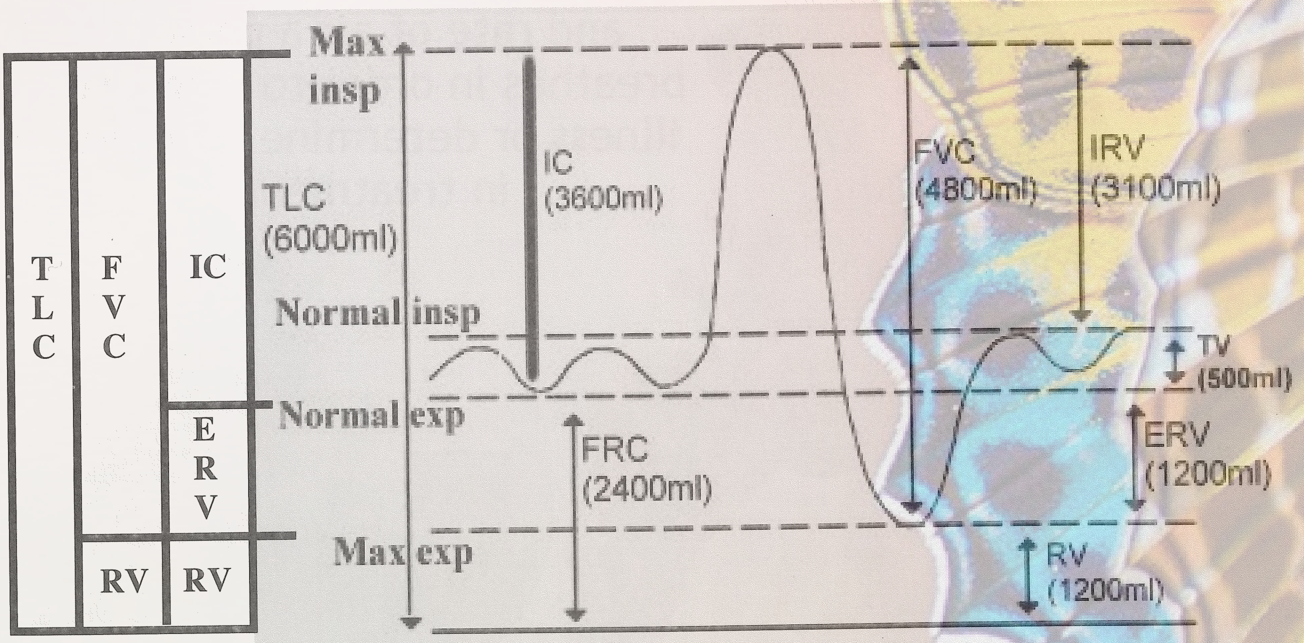
Inspiratory Capacity: IC

- It is the maximum volume of air that can be inhaled after a normal tidal breath out.
- Normally ~ 3600 ml (TV + IRV)

Maximum Breathing Capacity: MBC

- The maximum volume of air that can be respired in one min. by the deepest & fastest breathing.
- Normally, 120 L / min.

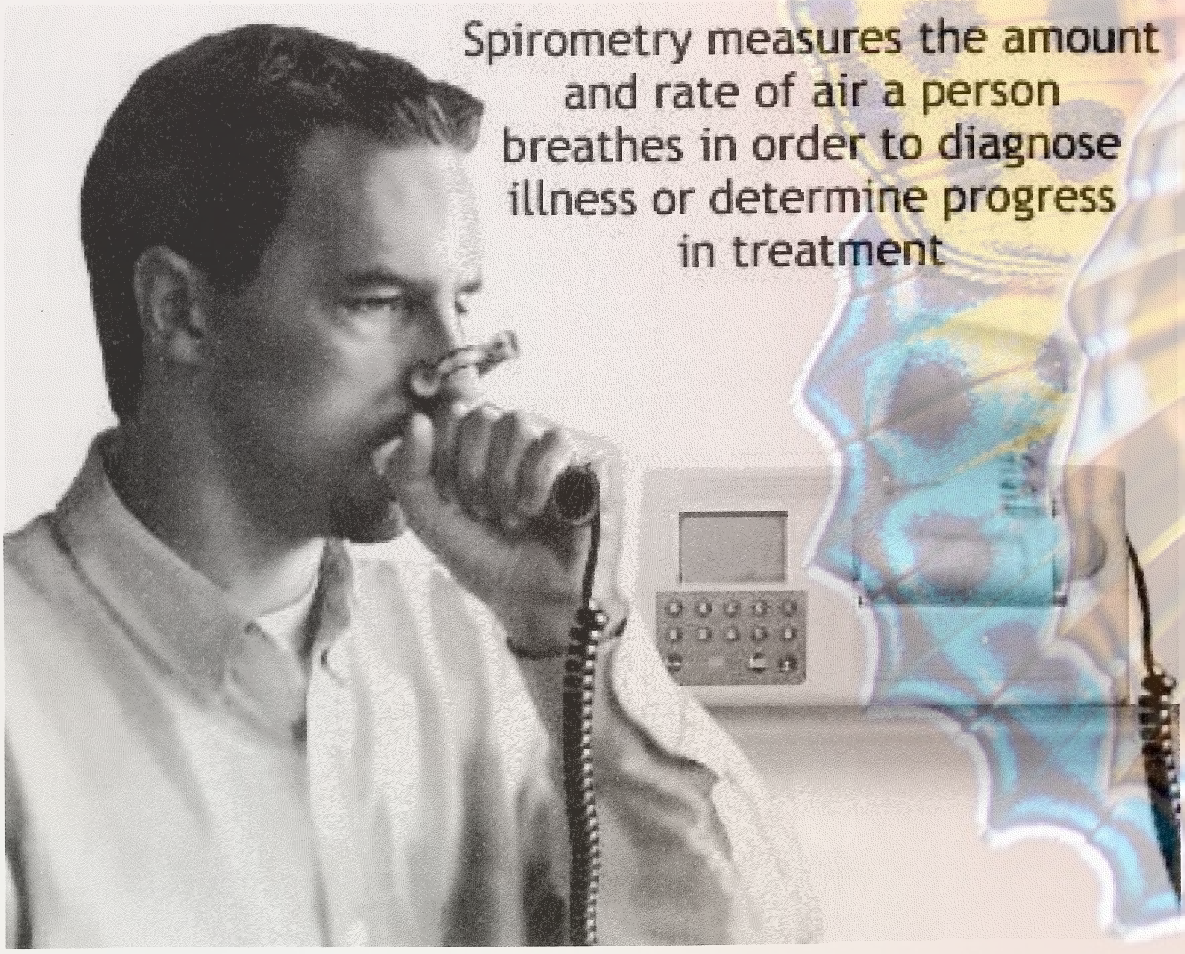
RESPIRATORY VOLUMES


T L C
F V C
IC
E R V
RV
RV
Max insp
TLC (6000ml)
IC (3600ml)
Normal insp
Normal exp
FRC (2400ml)
Max exp
FVC (4800ml)
IRV (3100ml)
TV (500ml)
ERV (1200ml)
RV (1200ml)

Lung Volumes measured with a spirometer during quiet breathing with one maximum breath. Values shown are for an average-sized healthy young male.

RV	*Residual volume*	*FRC*	*Functional residual capacity*
ERV	*Expiratory reserve volume*	*TV*	*Tidal vlume*
IRV	*Inspiratory reserve volume*	*FVC*	*Forced vital capacity*
TLC	*Total lung capacity*	*IC*	*Inspiratory capacity*

SPIROMETER



Spirometry measures the amount and rate of air a person breathes in order to diagnose illness or determine progress in treatment

Pattern of restrictive hypoventilation in RFTs

- Respiratory volumes & capacities:
  - Decreased: FVC.
  - Normal: RV.
  - Decreased: TLC.
- Expiratory flow rates:
  - Normal: FEV_1 / FVC.
- Decreased lung compliance.

Pattern of obstructive hypoventilation in RFTs

- Respiratory volumes & capacities:
  - Decreased: FVC.
  - Increased: RV.
  - Increased: TLC.
- Expiratory flow rates:
  - Decreased: FEV_1 / FVC.
- Increased lung compliance.

RESPIRATORY FAILURE

DEFINITION

- Laboratory diagnosis characterized by:
  - HYPOXIA (≤ 60 mm Hg) with or without:
  - HYPERCAPNEA (≥ 50 mm Hg),

due to a disease affecting respiration.

ETIOLOGY

1- Impaired diffusion.

2- Hypoventilation.

} See before

TYPES

A) According to the gas defect:

1. HYPOXEMIC TYPE (TYPE I): "mainly due to ↓ diffusion"
   - HYPOXIA: with normocapnia or hypocapnia.
2. HYPERCAPNIC TYPE (TYPE II): "mainly due to ↓ ventilation"
   - HYPOXIA: with HYPERCAPNIA.

B) According to the clinical onset:

1. ACUTE TYPE:
   - Onset: acute.
   - pH in Type II: markedly ↓↓ (respiratory acidosis).
2. CHRONIC TYPE:
   - Onset: gradual.
   - pH in Type II: slightly ↓↓ (renal compensation).

CLINICAL PICTURE

1- Manifestations of: HYPOXIA.

2- May be manifestations of: HYPERCAPNEA.

3- Manifestations of the cause: **e.g.**

- Features of *COPD:* chronic respiratory failure.
- Features of *GBS:* acute respiratory failure.

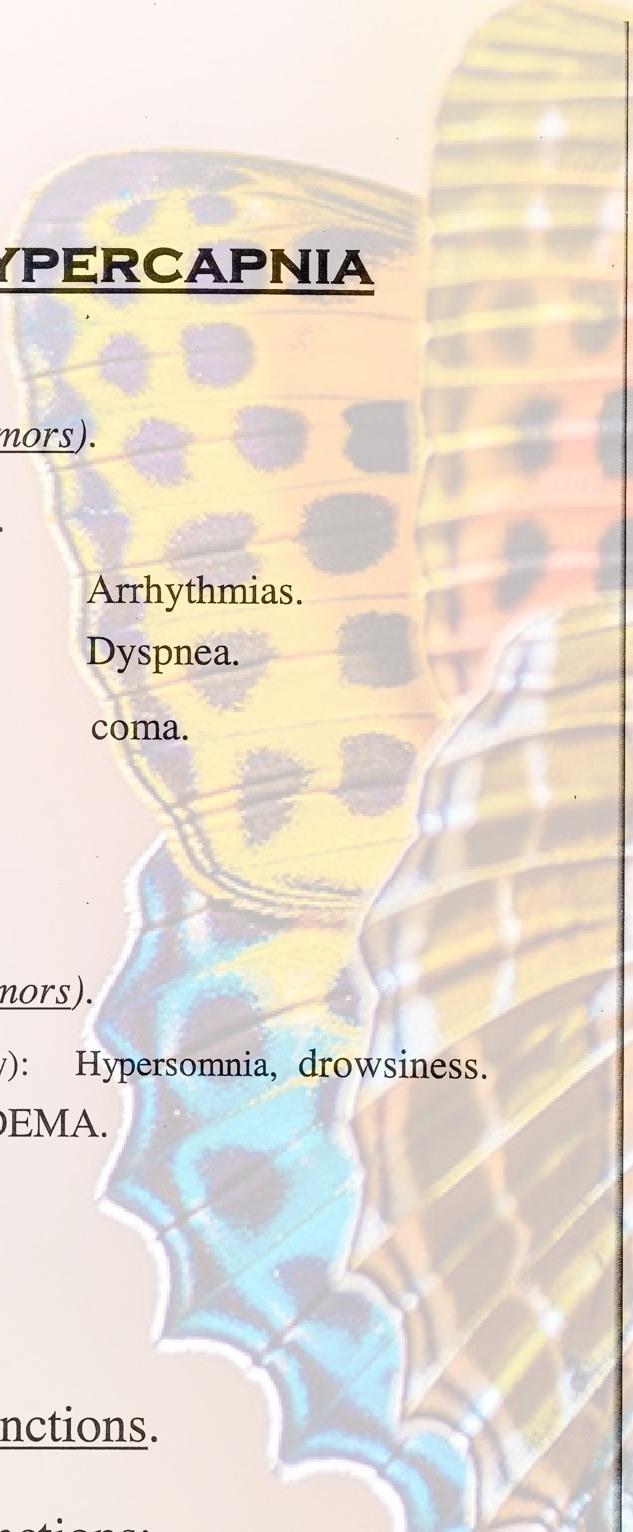
MANIFESTATIONS OF HYPOXIA

1. Acute hypoxia:

- BP: Hypotension.
- PULSE: Tachycardia, Arrhythmias.
- RESPIRATION: Tachypnea, Dyspnea.
- **C**entral **C**yanosis.
- **D**ROWSINESS: convulsions, coma.
- **D**EATH.

2. Chronic hypoxia:

- Headache.
- Polycythemia (***secondary***).
- **C**or pulmonale (***secondary to pulmonary hypertension***).
- **C**entral **C**yanosis.
- **C**lubbing.
- Apathy, fatigue, drowsiness.



MANIFESTATIONS OF HYPERCAPNIA

1. Acute hypercapnia:

- **A**sterixis (*flapping tremors*).
- **A**cidosis (*respiratory*).
- PULSE: Tachycardia, Arrhythmias.
- RESPIRATION: Tachypnea, Dyspnea.
- **D**ROWSINESS: convulsions, coma.
- **D**EATH.

2. Chronic hypercapnia:

- **A**sterixis (*flapping tremors*).
- CO_2 narcosis (Hypercapnic encephalopathy): Hypersomnia, drowsiness.
- INCREASED ICT, PAPILLOEDEMA.

INVESTIGATIONS

1- Tests for specific pulmonary functions.

2- Tests for general pulmonary functions:

a. ABG: (PO_2 , PCO_2) & (pH , HCO_3).
b. Pulse Oximetry.

3- Investigations of the cause: **e.g.**

- Investigations of *COPD:* chronic respiratory failure.
- Investigations of *GBS:* acute respiratory failure.

TREATMENT

1- Admission to hospital: *in a respiratory ICU*.

2- Supportive treatment:

A) *Improvement of ventilation:*

1. Maintenance of patent airways.
2. Elimination of retained secretions:
    - Encourage the patient to cough.
    - Respiratory exercises.
    - Expectorants **e.g.** *potassium iodide*.
    - Liquefaction of suptum:
        - Adequate hydration.
        - Mucolytics **e.g.** *bromehexine*.
    - Suction of secretions through:
        - Catheters.
        - Endotracheal tube.

B) *Oxygen inhalation.*

C) *Mechanical ventilation.*

3- Treatment of the cause: **e.g.**

- Treatment of *COPD:* chronic respiratory failure.
- Treatment of *GBS:* acute respiratory failure.

4- Treatment of complications: **e.g.**

- Treatment of *pulmonary infections*.
- Treatment of *acidosis*: (in acute Type II RF).

CHRONIC BRONCHITIS

DEFINITION

A condition characterized by ***excessive mucus secretion*** from the bronchial tree, in which there is productive cough every day, or most days, for ***at least 3 months*** of the year, for at least ***2 successive years***.

ETIOLOGY & PREDISPOSING FACTORS

(7 S)

1. **S**ex: males more than females.
2. **S**usceptibility: family history common.
3. **S**moking: (*the most common cause*).
4. **S**mog: atmospheric pollution.

} **Bronchial irritation**

5. **S**inusitis: chronic sinusitis predisposes to bronchitis.
6. **S**uperinfection: St. pneumoniae & H. influenza.
7. **S**ocial class: lower socio economic groups than higher.
8. OTHERS:
    - a. Chronic lung congestion (*LVF*).
    - b. Chronic lung plethora (*congenital heart disease with Lt to Rt shunt*).
    - c. Bronchiectasis.
    - d. Allergic factors: may play a role.

PATHOLOGY

- There are **3 types** which represent gradual increase in the severity of the disease:

1. *Chronic simple bronchitis*:

- There is hypertrophy of the mucus-secreting glands.
- The patient expectorates whitish mucoid sputum.

2. *Chronic mucopurulent bronchitis*:

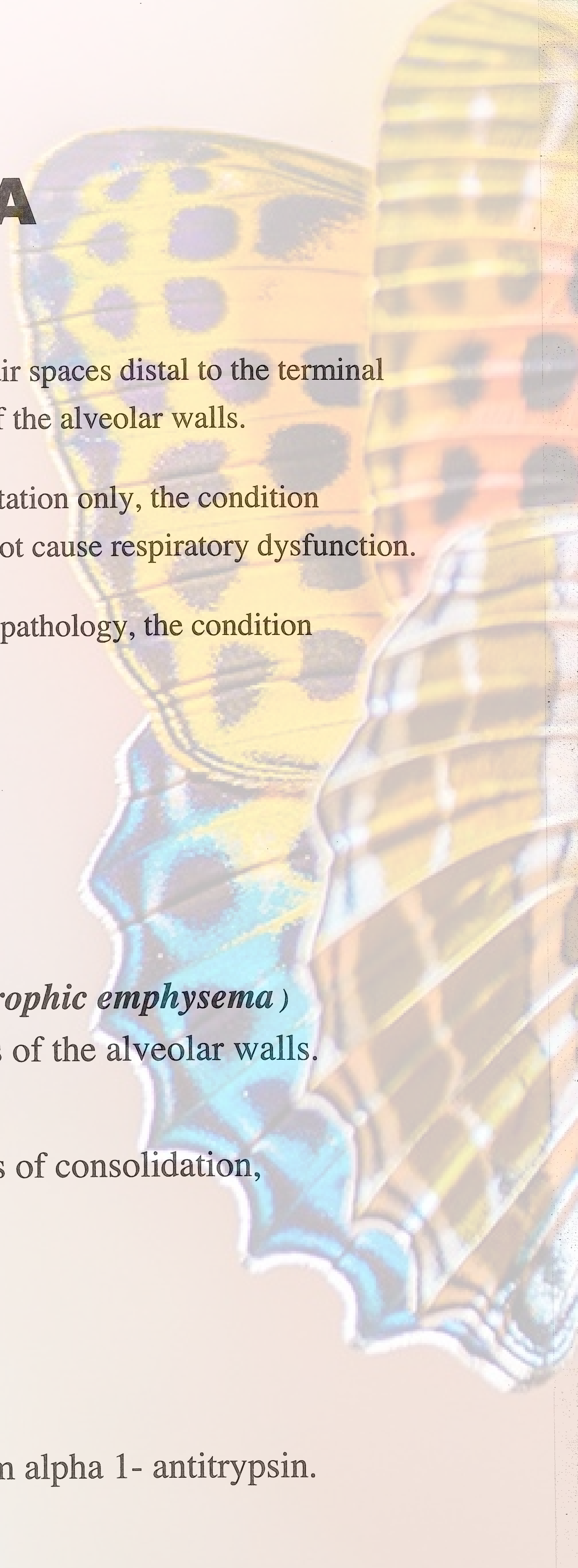
- There is secondary infection most commonly with:
  St. pneum. & H. influenzae.

- The patient expectorates yellowish mucopurulent sputum.

3. *Chronic obstructive bronchitis*:

- There is chronic irreversible airway obstruction, mainly affecting the small airways, due to:
  - Thickening of the bronchial wall.
  - Peribronchial fibrosis.

- This condition may be complicated by emphysema.



EMPHYSEMA

DEFINITION

- A **pathologic term** indicating enlargement of the air spaces distal to the terminal bronchioles due to dilatation and / or destruction of the alveolar walls.

- When the expansion of the air spaces is due to dilatation only, the condition may be termed " false emphysema " & it does not cause respiratory dysfunction.

- When destruction of the alveolar walls is the main pathology, the condition is termed: " true emhysema."

ETIOLOGY

I. False emphysema:

1. **Senile empysema:** *(atrophic emphysema)*
   - Occurs in old age due to weakness of the alveolar walls.

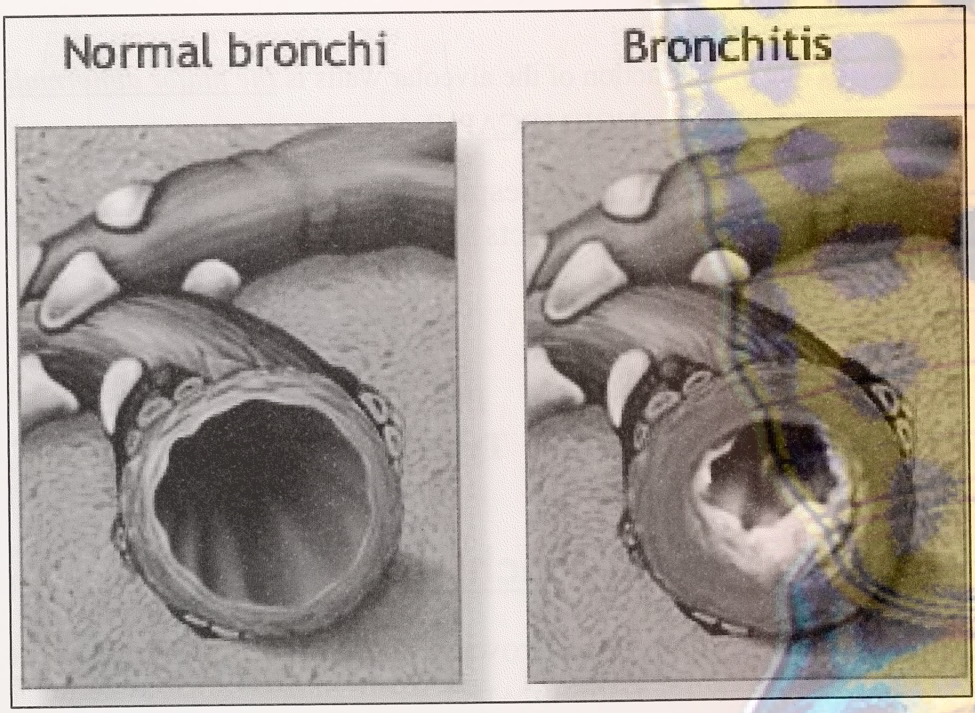
2. **Compensatory emphysema:**
   - Occurs in lobules adjacent to areas of consolidation, collapse, fibrosis or resection.

II. True emphysema:

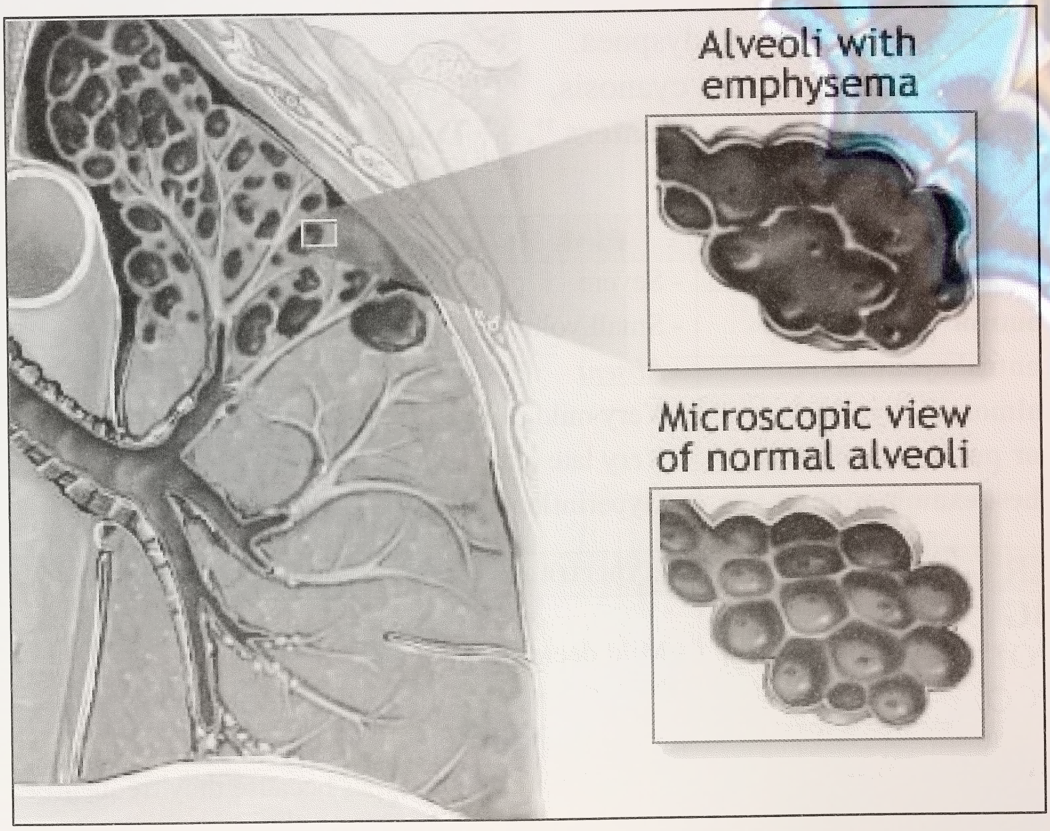
1. **Primary emphysema:**
   - Occurs due to deficiency of serum alpha 1- antitrypsin.

2. **Secondary emphysema:**
   - This is the most important type & occurs as a complication of:
     - Chronic obstructive bronchitis *(the most common cause)*.
     - Bronchial asthma.

BRONCHITIS


Normal bronchi
Bronchitis

EMPHYSEMA


Alveoli with emphysema
Microscopic view of normal alveoli

EMPHYSEMA versus CHRONIC OBSTRUCTIVE BRONCHITIS

- In emphysema:

- ***Impaired diffusion*** due to destruction of the alveolar walls is ***the major abnormality*** leading to:
  HYPOXIA & NORMOCAPNEA.
- Due to HYPOXIA, stimulation of the respiratory center occurs & results in: severe DYSPNEA which partially corrects the hypoxia.
- ***Conclusion:***
  - Presence of: severe dyspnea.
  - Absence of: Central cyanosis, PH, corpulmonale, RVF.
  - This type is: **" Pink puffers ", Type A, or Emphysematous Type.**

- In chronic obstructive bronchitis:

- ***Impaired ventilation*** due to airway narrowing is ***the major abnormality*** leading to:
  HYPOXIA & HYPERCAPNEA.
- Due to UNKNOWN REASON, stimulation of the respiratory center does not occur, this results in: absence of severe DYSPNEA.
- ***Conclusion:***
  - Absence of: severe dyspnea.
  - Presence of: Central cyanosis, PH, corpulmonale, RVF.
  - This type is: **" Blue Bloaters ", Type B, or Bronchitic Type.**

	Pink Puffers	**Blue Bloaters**
- Dyspnea	- Severe	- Very mild
- Sputum	- Small volume	- Large volume
- Central cyanosis	- *Absent*	- *Present*
- Pulmonary hypertension	- Very mild or *absent*	- *Present*
- Cor pulmonale & RVF	- Very late or *absent*	- *Present*
- Chest examination	- Hyperinflation	- Wheezes
CXR	**- Hypertranslucency**	**- No Hypertranslucency**
ABG		
- PO_2	- Mild decrease	- Severe decrease
- PCO_2	- Normal	- High

HOWEVER: there is usually overlap between both types because they frequently coexist. So, it is difficult to differentiate them into 2 separate clinical types.

Chronic Obstructive Pulmonary Disease
COPD

DEFINITION

- A group of chronic & slowly progressive respiratory disorders characterized by: ***reduced expiratory flow rates.***
- It includes: ***chronic obstructive bronchitis***, & ***emphysema*** which usually co-exist.

PATHOPHYSIOLOGY

1. Impaired ventilation: *" obstructive hypoventilation "*

- This is due to: narrowing of the airways.

2. Impaired diffusion:

- Destruction of the alveolar walls causes: diminished surface area of diffusion.

3. Impaired perfusion:

- Destruction of the alveolar septa causes: loss of the capillaries.

4. Hyperinflation:

- It is due to: increased residual volume due to obstruction.
- It leads to: signs of hyperinflated chest.

5. Cardiovascular abnormalities:

I. Pulmonary hypertension: due to:

1. Hypoxia: will cause:
    - Direct VC of the pulmonary arteries.
    - Increased blood viscosity due to polycythemia.
2. Reduction of the pulmonary vascular bed: due to:
    - Loss of the capillaries: due to destruction of the alveolar septa.
    - Compression of the capillaries: due to ↑ intra – alveolar pressure.

II. Hypoxic cor pulmonale.

6. **Renal & hormonal abnormalities:**

- Chronic hypoxia & hypercapnea have been shown to cause increased levels of:
  renin, ***aldosterone***, ***noradrenaline***.

- Salt & water retention will occur: even in the absence of RVF.

CLINICAL PICTURE

Type of patient

- Usually a male, chronic heavy smoker, above 50 years.

Symptoms

1. **Long history of chronic bronchitis:**

- Chronic cough: with expectoration of mucoid sputum.
- Chronic cough: with expectoration of mucopurulent sputum.
- Chronic cough: with gradual progressive dyspnea & wheezing.

2. **Chest pain,** due to:

- Pain in intercostals muscles.. from chronic cough.
- Pneumothorax……………… from rupture of emphysematous bullae.
- Pleurisy ……………………… from complicating pneumonia.

3. **Oedema of lower limbs,** due to:

- RVF.
- Salt & water retention.

4. **Symptoms of complications,** e.g. *respiratory failure*.

Signs

GENERAL SIGNS

1. Vital signs:

PULSE:

A) Volume:

In early cases:

- Big pulse volume: due to VD effect of hypoxia.

In late cases:

- Small pulse volume: due to PH & RVF.

In severe cases:

- Pulsus paradoxus.

B) Rhythm:

Arrhythmias: *especially*, MAT, AF.

RESPIRATORY RATE:

Tachypnea: *with working accessory respiratory muscles*.

2. Head examination:

- Puffiness of the eye lids: due to chronic cough.
- Central cyanosis: may be present.

3. Neck examination: "Congested pulsating"

- Increased intrathoracic pressure.
- RVF.

4. Upper limbs:

- Mild clubbing: *is the rule*.
- Marked clubbing: *occurs if there is* ***associated bronchiectasis***.

5. Lower limbs: "Oedema"

- RVF.
- Salt & water retention.

6. Abdomen:

1. Liver:

- The lower border of the liver may be palpable due to:
  - a) *Ptosed liver:* due to hyperinflation ***(non tender).***
  - b) *Enlarged liver:* due to RVF ***(tender).***

2. Ascites:

- It is due to RVF.

7. General signs of complications, e.g. respiratory failure, *especially:*

- **C**entral **C**yanosis.
- **A**sterixis *(flapping tremors).*
- CO$_2$ narcosis (Hypercapnic encephalopathy): Hypersomnia, drowsiness.

CHEST SIGNS

Inspection

- Shape: barrel shaped chest.
- Movement: decreased bilaterally.

Palpation

- **T**rachea: central.
- **T**VF: decreased bilaterally all over the chest.

Percussion

- Hyperresonance: all ovet the chest.
- Encroachement on: the normal hepatic & cardiac dullness.

Auscultation

- Breath sounds: vesicular with prolonged expiration.
- Additional sounds: generalized rhonchi, mainly expiratory, may be early inspiratory crepitations.

COMPLICATIONS

1. Respiratory failure.
2. Right sided heart failure following cor pulmonale.
3. Left sided heart failure.

Failure

4. Pulmonary infections.
5. Pneumothorax.
6. Bronchiectasis.

Chest complications

7. Polycythemia.
8. Salt & water retention.
9. Complications of chronic cough.

General complications

Causes of Left sided HF in COPD

1. Hypoxia: leading to myocardial ***ischemia***.
2. Acidosis: leading to myocardial ***depression***.
3. Volume overload due to: hyperdynamic circulation due to VD.
4. Increased blood viscosity due to: polycythemia.
5. Reversed Bernheim effect.
6. Associated disease of the left side of the heart, **especially CAD**.

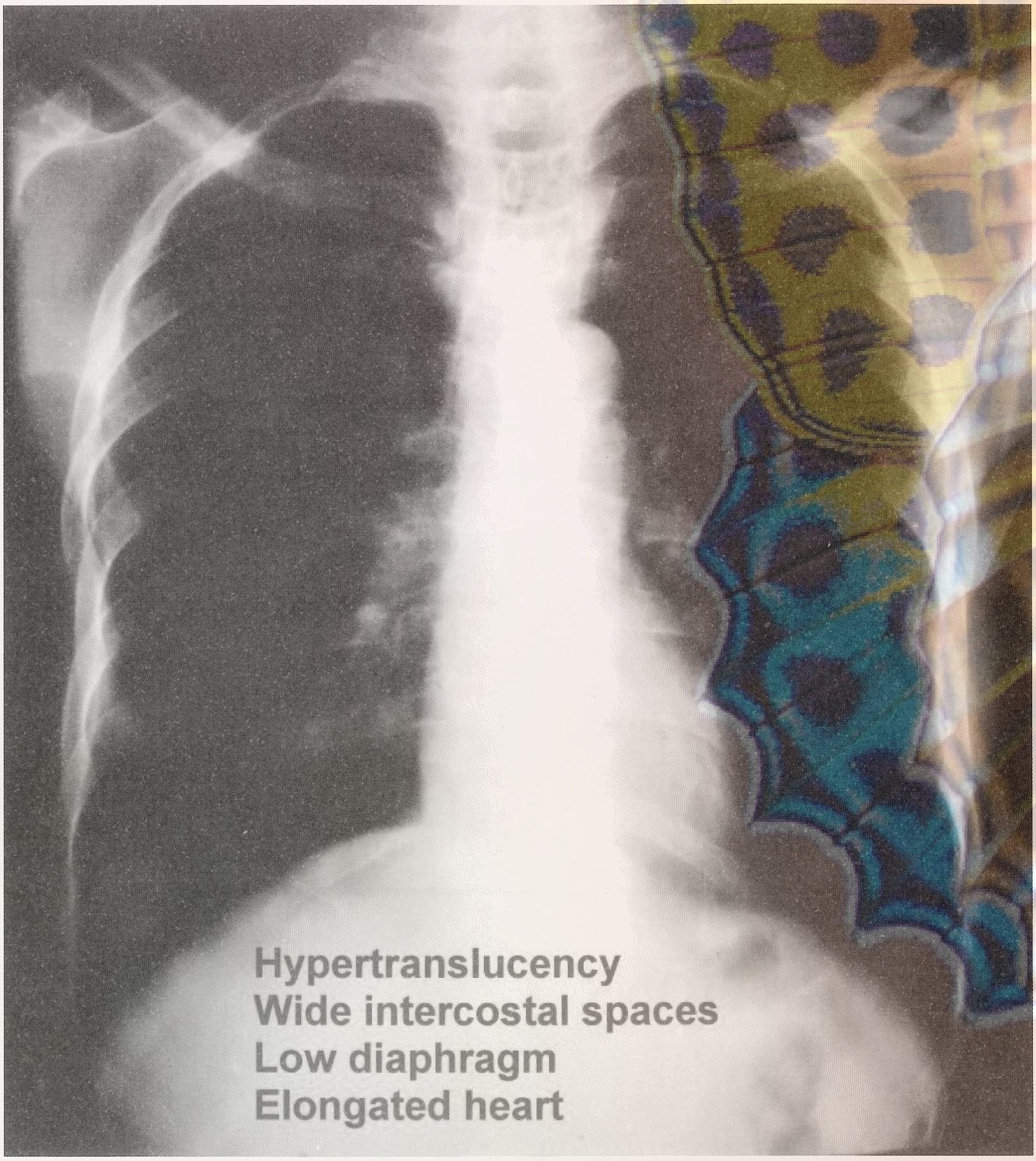
INVESTIGATIONS

1. CXR:

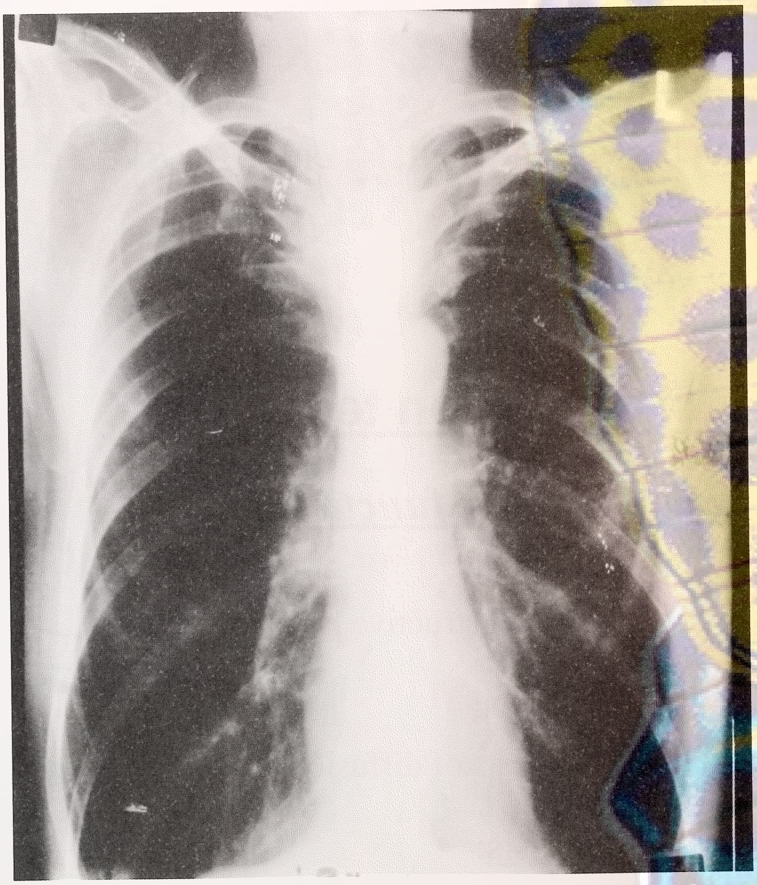
a) *Signs of hyperinflation*:

- Hypertranslucency of the lung fields.
- Wide intercostal spaces.
- Low flat diaphragm.
- Heart shadow is elongated " *ribbon-shaped heart* ".
- May be emphysematous bullae.

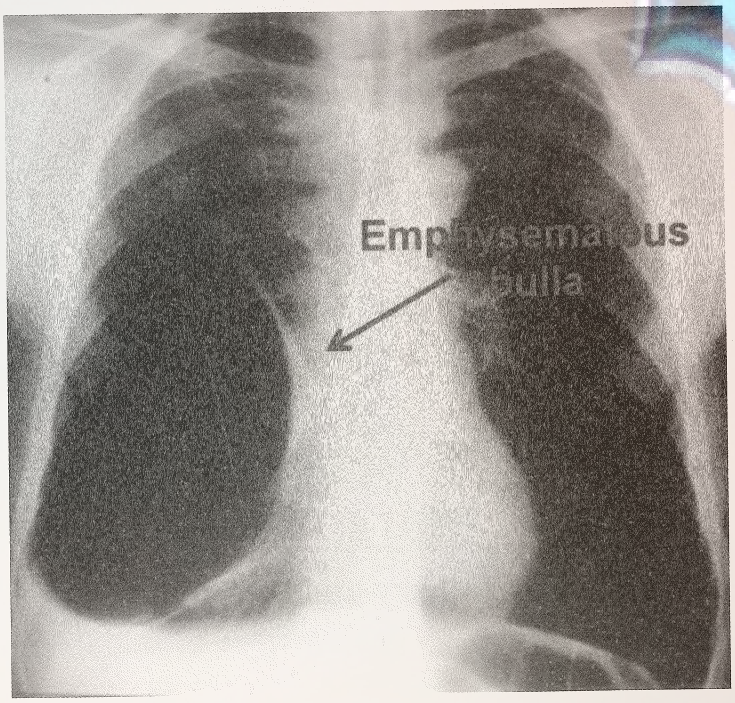
b) *Increased bronchovascular markings*.



Hypertranslucency
Wide intercostal spaces
Low diaphragm
Elongated heart



Low diaphragm, Elongated heart, ↑ bronchovascular markings


Emphysematous
bulla

2. ECG:

- Arrhythmias, *e.g.* *(MAT, AF).*
- Chamber enlargement, *e.g.* *(RV++ & RA++).*
- CAD.
- Low voltage.

3. Respiratory function tests:

Specific pulmonary function tests:

a) Ventilation tests:
- Features of obstructive hypoventilation.

b) Diffusion tests:
- Disturbed tests of diffusion: *decreased CO transfer factor*.

c) Perfusion tests:
- Disturbed tests of perfusion.

General pulmonary function tests: **(ABG)**

- Hypoxia.
- Hypercapnea.
- Increased bicarbonate.

4. Blood picture:

- May show: secondary polycythemia.

5. Serum alpha 1-antitrypsin:

- Decreased in primary emphysema.

TREATMENT

1. Avoid further bronchial irritation:

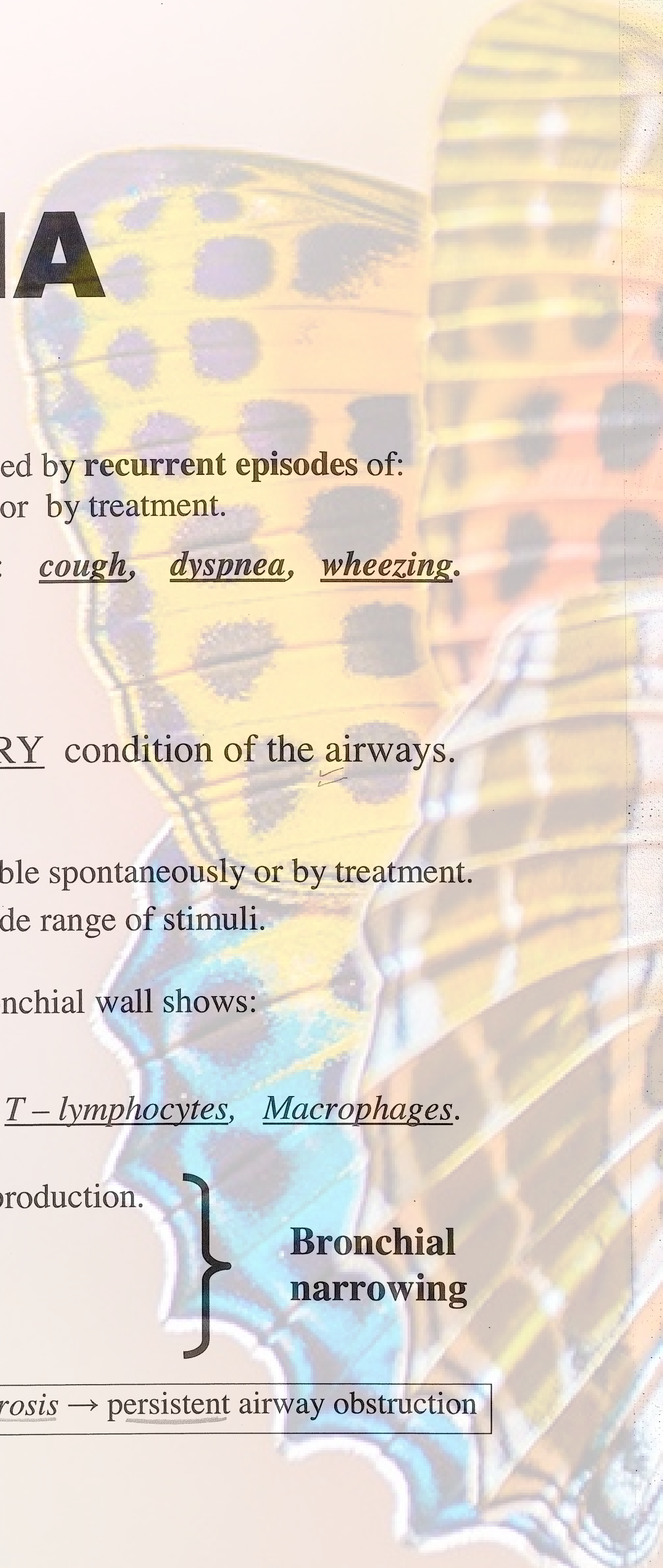
- Stop smoking.
- Avoid atmospheric pollution.

2. Treatment of complications:

- **e.g.** respiratory infections, antibiotics according to: C & S.
- **e.g.** respiratory failure.

3. Symptomatic treatment:

- Expectorants, *e.g.* potassium iodide.
- Mucolytics, *e.g.* bromhexine.
- Bronchodilators, *e.g.* β_2-stimulants.
- Corticosteroids, *e.g.* prednisone 1 mg / Kg orally daily in:
    - *Patients not responding to bronchodilators.*
- OXYGEN THERAPY.



ASTHMA

DEFINITION

- It is a *CLINICAL SYNDROME* characterized by **recurrent episodes** of: **airway obstruction** that resolve spontaneously or by treatment.
- The clinical episodes include 3 main symptoms: ***cough***, ***dyspnea***, ***wheezing***.

PATHOLOGY

- It is a common chronic INFLAMMATORY condition of the airways.
- It has 3 main characteristics:
  1. Airflow **limitation:** reversible spontaneously or by treatment.
  2. Airway **hyper-responsiveness:** to a wide range of stimuli.
  3. Airway **INFLAMMATION:** the bronchial wall shows:
     - Epithelial damage.
     - Cells: *Eosinophils*, *Mast cells*, *T – lymphocytes*, *Macrophages*.
     - Goblet cell hyperplasia → ↑ mucus production.
     - Oedema.
     - Smooth muscle hypertrophy.

Bronchial narrowing

With time, chronic inflammation → *submucosal fibrosis* → persistent airway obstruction

CLASSIFICATION

1. Extrinsic asthma (Allergic asthma):

- There is a definite external cause, especially *antigen* or *allergen*.
- It occurs in *ATOPIC* individuals.

2. Intrinsic asthma (Idiosyncratic):

- There is no definite external cause, no *antigen* no *allergen*.
- Non – antigenic stimuli may be responsible.

Patients commonly show features of both types of asthma.

ETIOLOGY & PATHOGENESIS

I. Extrinsic asthma

(atopy is the major factor)

1. It occurs in atopic individuals:

- It runs in families.
- Associated atopic diseases: e.g. allergic rhinitis.
- Early – onset asthma: starts usually in childhood.
- Skin tests: positive.
- Serum IgE level: increased.

2. It occurs due to exposure to allergic stimuli:

- **In**haled allergens, e.g. house dust.
- **In**gested allergens, e.g. fish, eggs.
- **In**jected allergens, e.g. penicillin.
- Contact -

3. MECHANISM:

a. **S**ensitization:

- When allergens are inhaled for the first time, specific **IgE antibodies** are formed and bind to receptors on the surface of **Mast cells** of the tracheo-bronchial tree.

b. **S**timulation: (initial response)

- When these allergens are inhaled for the second time & repeatedly, they will bind to IgE antibodies → stimulation of mast cells.
- Mast cells will degenerate → release of **Mediators** → asthma.

c. **S**ignaling: (secondary response = late-onset response)

- Following stimulation of mast cells, these cells begin to signal other cells (lymphocytes, macrophages & epithelial cells) → upregulation of these cells, extension of inflammation & more release of mediators.

d. Migration: of eosinophils into the airways occurs under the effect of chemotactic cytokines.

e. Activation: of eosinophils under the effect of cytokines (IL-5) → release of:

- Toxic products (e.g. Major basic protein).
- Mediators (e.g. Leukotrienes & PAF).

f. Epithelial damage: occurs under the effect of eosinophil products.

II. Intrinsic asthma *(airway hyper-responsiveness is the major factor)*

1. It is not related to atopy or allergy:

- It does not run in families.
- No associated atopic diseases.
- Late – onset asthma: starts usually in middle age.
- Skin tests: negative.
- Serum IgE level: normal.

2. It occurs due to exposure to non – allergic stimuli (triggers):

- **En**vironmental: e.g. pollution & industrial materials.
- **E***xposure to*: respiratory tract infections (*esp. viruses*).
- **E***xposure to*: cold air.
- **E**xercise: **exercise – induced asthma**.
- **E**motional stress.
- **DRUGS:** **A**spirin (& other NSAIDs), **ß**-blockers.

3. MECHANISM:

- Probably these patients have **highly sensitive Vagal receptors** in the tracheo-bronchial tree.
- **Non-antigenic stimulation** of these receptors results in:
  - **Re**flex bronchospasm.
  - **S**timulation: of **Mast cells** → release of **Mediators.**
  - **S**ignaling: as mentioned above.
  - Migration: as mentioned above.
  - Activation: as mentioned above.
  - Epithelial damage: as mentioned above.

Mediators involved in asthma

- Histam*ine*.
- Adenos*ine*.
- Seroton*in*.
- Prostagland*ins*.
- Kin*ins*.
- LEUKOTRIENES.
- CYTOKINES.
- PAF (Platelet activating factor).

CLINICAL PICTURE

I. ATTACKS OF ASTHMA:

Symptoms “ Attacks ”

- **Timing:**
  - Usually occur in late night & early morning due to **High vagal tone**.

- **Description:**
  - Typically: a **Triad** of, Dyspnea, cough, wheezing.
  - Cough-variant asthma: cough (esp. nocturnal) may be the only presenting C/O.
  - Expectoration: viscid sputum (mucous pellets).
  - Association: anxiety, sweating.

- **Duration:** (Variable)
  - Few ***minutes*** ………… to several ***hours*** ……………or even ***days***.
  - Resolve spontaneously or by treatment.

Signs

1. **During the attack:** “ Signs of Hyperinflation & Airway obstruction”

Inspection

- Shape: barrel shaped chest.
- Movement: decreased bilaterally.

Palpation

- **T**rachea: central.
- **T**VF: decreased bilaterally.

Percussion

- Hyperresonance.

Auscultation

- Breath sounds: vesicular with prolonged expiration.
- Additional sounds: generalized rhonchi, mainly expiratory.

2. **In between the attacks:**

- The chest is usually free, unless:
  - Bronchial asthma is accompanied with chronic bronchitis.
  - Submucosal fibrosis occurs in chronic cases → persistent obstruction.

II. CLASSIFICATION OF ASTHMA:

Asthma severity is divided into the same classes in the:

NAEPP [1], and **GINA** [2] guidelines.

Classification is based on:

1. History of asthma symptoms.
2. Lung functions.

} **Without ttt**

NAEPP / GINA Classification of Asthma Severity

	Symptoms/Day	Symptoms/Night	PEFR or FEV$_1$
STEP 1 ***Mild Intermittent***	≤ 2 times a week No symptoms between attacks	≤ 2 times a month	≥ 80%
STEP 2 ***Mild Persistent***	> 2 times a week but < 1 time a day Attacks may affect daily activity	> 2 times a month	≥ 80%
STEP 3 ***Moderate Persistent***	Daily Attacks affect daily activity	> 1 time a week	60% - 80%
STEP 4 ***Severe Persistent***	Continuous Severe limitation of daily activity	Frequent	≤ 60%

- An asthmatic patient belongs to a certain class if:
  [*ONLY* one feature of that class is present ***"without treatment"***].

---

[1] **NAEPP**: The **N**ational **A**sthma **E**ducation and **P**revention **P**rogram

[2] **GINA**: The **G**lobal **In**itiative for **A**sthma

III. STATUS ASTHMATICUS:

(acute severe asthma)

- It is a medical emergency:
  The severest form of asthma that remains unresponsive to the standard treatment of asthma for several hours.
- It is of variable severity:
  It may be either: *severe* or *life – threatening*.

Manifestations of severe asthma:

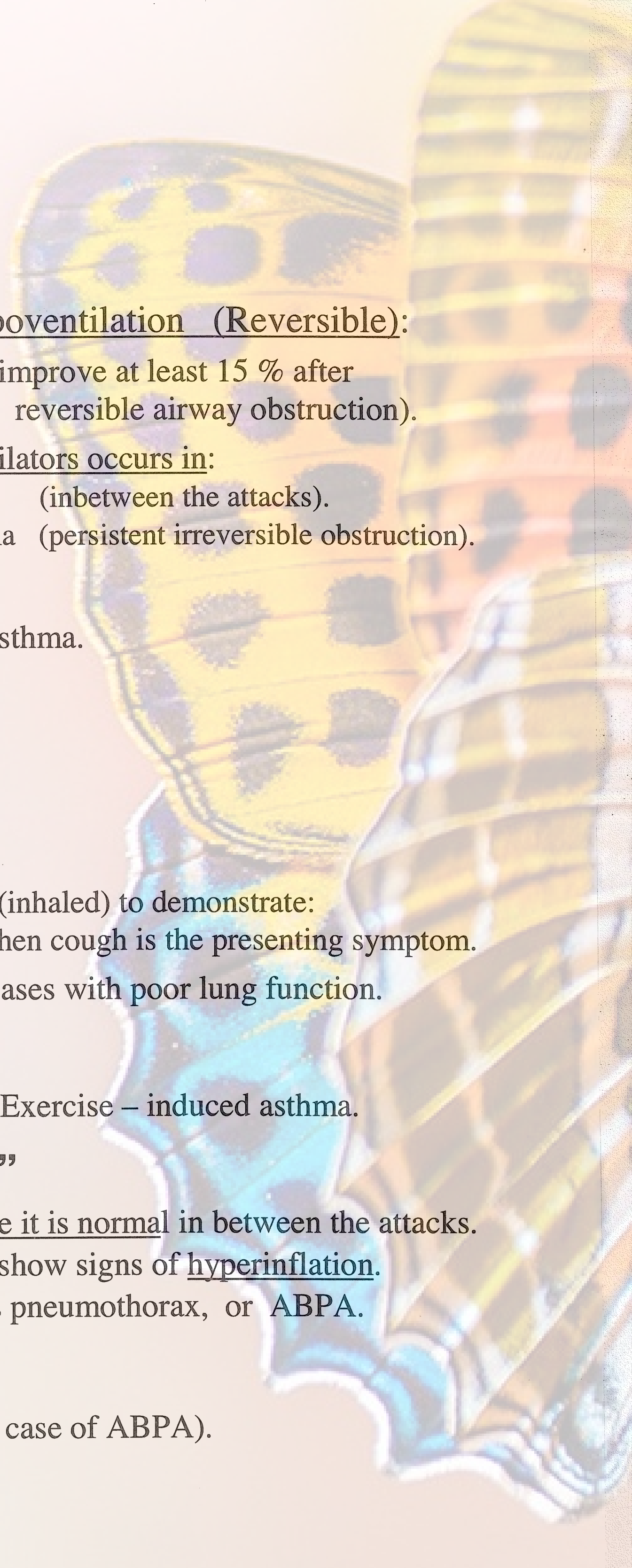
- RESPIRATION:
  - Inability to complete a sentence in one breath.
  - Tachypnea: RR > 25 breaths per minute.
- PULSE:
  - Pulsus paradoxus.
  - Tachycardia: HR > 110 beats per minute.
- RESPIRATORY FUNCTIONS:
  - Marked decrease of: PEFR & FEV_1 (< 50% of normal).

Manifestations of life threatening asthma:

- Silent chest.
- Central cyanosis.
- Confusion or coma.
- RESPIRATORY FUNCTIONS:
  - Marked decrease of: PEFR & FEV_1 (< 30% of normal).
- ARTERIAL BLOOD GASES (ABG):
  - Marked hypoxemia: PO_2 < 60 mmHg (despite ttt with O_2).
  - Marked hypercapnea: PCO_2 > 45 mmHg.
  - Low or falling pH.

COMPLICATIONS

1. STATUS ASTHMATICUS.
2. COPD.
3. Respiratory *failure*.
4. Right sided heart *failure* & corpulmonale.
5. Complications of: severe cough.
6. Complications of: therapy e.g. *corticosteroids*.
7. Pneumothorax & pneumomediastinum.
8. Allergic bronchopulmonary aspergillosis (ABPA).



INVESTIGATIONS

1. Pulmonary function **tests**:

I. VENTILATION: Obstructive hypoventilation (Reversible):

- Decreased PEFR & FEV_1 which improve at least 15 % after inhalation of a bronchodilator (i.e. reversible airway obstruction).
- Absent response to inhaled bronchodilators occurs in:
  - a) Asthma in remission (inbetween the attacks).
  - b) Severe longstanding chronic asthma (persistent irreversible obstruction).

II. DIFFUSION:

- CO transfer factor: normal in asthma.

III. Arterial blood gases (ABG):

- PO_2: Decreased.
- PCO_2: Increased.

2. Bronchial provocation **tests**:

- These tests use Histamine or metacholine (inhaled) to demonstrate: ***airway hyper-responsiveness*** especially when cough is the presenting symptom.
- These tests are contraindicated in severe cases with poor lung function.

3. Exercise **tests**:

- These tests use Treadmill to diagnose: Exercise – induced asthma.

4. **CXR**: " ***little role*** "

- Little role in diagnosing asthma: because it is normal in between the attacks.
- During the attack: it may show signs of hyperinflation.
- May show complications: such as pneumothorax, or ABPA.

5. **CBC**:

- Excessive eosinophils (more marked in case of ABPA).

6. **Sputum examination**:

- Excessive eosinophils.
- *Curshman's spirals.*
- *Charcot – Leyden crystals.*
- *Creola bodies.*

7. **Skin tests**:

- To identify the allergen in extrinsic asthma.

8. **Serum IgE**:

- Increased in most cases of extrinsic asthma.

DIFFERENTIAL DIAGNOSIS

1. ***From other causes of paroxysmal dyspnea with wheezing*:**
    - Cardiac asthma.
    - Uremic asthma.
    - Carcinoid syndrome.
    - Pulmonary vasculitis.

2. ***From other causes of paroxysmal dyspnea*:**
    - Cardiac dyspnea.
    - Extrinsic allergic alveolitis.
    - Tetany (laryngismus stridulus).
    - Myasthenic crisis.
    - Recurrent pulmonary emboli.

3. ***From chronic bronchitis.***

4. ***Differentiate between:*** ***extrinsic & intrinsic asthma.***

TREATMENT

1 GENERAL MEASURES

- Avoid exposure to stimuli: whether ***allergic*** or ***non –allergic***.
- Treatment of: CHEST INFECTIONS.
- PSYCHOTHERAPY.
- **IMMUNOTHERAPY: (HYPOSENSITIZATION)**
    - *Repeated injection of extracts of the allergen in gradually increasing doses may be beneficial……… **why** ??*
    - *It results in production of IgG antibodies which will prevent binding of the allergen to IgE (**blocking antibodies**).*

2 SPECIFIC MEASURES (DRUGS)

1. BRONCHODILATORS

A) *Sympathomimetics:*

1. **Selective β2-Agonists:** [INHALED]

   THEY HAVE A RAPID ONSET OF ACTION & FEW SIDE EFFECTS.
   THEY COULD BE GIVEN PARENTRALLY IN STATUS ASTHMATICUS.

   - Salbutomol: (puff = 100 μg, 2 puffs tds).
   - Terbutaline: (puff = 250 μg, 2 puffs tds).
   - Salmetrol: (puff = 50 μg, 1 puff bid) .... LONG ACTING.

2. **Non Selective β2- Agonists:**

   THEY ARE OF LITTLE USE BECAUSE OF THEIR SIDE EFFECTS.

   - Adrenaline: 0.5 c.c. 1 / 1000 solution SC.
   - Isoprenaline: 10 mg SL or by inhalation.
   - Ephedrine: 30 mg orally.

B) *Xanthines:* (Theophylline or Aminophylline)

- Short acting: slow IV infusion in status asthmaticus.
- long acting: orally for long – term control.

2. RECEPTOR ANTAGONISTS

A) *Anticholinergic bronchodilators:* " Acetylcholine antagonists "

- Ipratropium bromide inhaler (20 - 40 μg tds).
- Oxitropium bromide inhaler.

B) *Antihistamines:* " H_1 receptor antagonists "

- Terfenadine (rarely used).

C) *Antileukotrienes:*

- Montelukast (Leukotriene receptor antagonist).
- Zileuton (Leukotriene synthesis inhibitor).

Monotherapy in *mild persistent asthma*, Add – on therapy in *moderate to severe asthma.*
Useful in: *aspirin – induced asthma.*

3. ANTI – INFLAMMATORY DRUGS

A) Corticosteroids:

1. Inhaled corticosteroids:

DRUG:

- Beclometha***sone*** (daily dose = see later).
- Flutica***sone***.
- Budesonide.

SIDE EFFECTS:

- Hoarseness of voice.
- Oropharyngeal candidiasis.

2. Oral corticosteroids:

DRUG:

- Predninsone: 1 mg Kg / day.

SIDE EFFECTS:

- Refer to: Rheumatoid arthritis.

3. Parentral corticosteroids: *(for Status asthmaticus)*

DRUG:

- Methylprednisolone.
- Hydrocortisone.

SIDE EFFECTS:

- Refer to: Rheumatoid arthritis.

B) Other anti-inflammatory drugs:

- Gold, Methotrexate, Cyclosporine.
- Under trial for severe chronic asthma not responding to other drugs.

4. PROPHYLACTIC DRUGS

A) Disodium cromoglycate:

- It inhibits release of mediators from mast cells (mast cell stabilizer).
- Dose: inhaled puff = 1 mg, 2 puffs / 6 hours.

B) Kitotifen:

- It inhibits release of mediators from mast cells (mast cell stabilizer).
- It has antihistaminic effect.
- Dose: oral tablet = 1 mg, 1 tablet bid.

C) Omalizumab: (Recombinant Anti – IgE)

- It combines with free IgE blocking its interaction with mast cells.
- Dose: single injection, (SC every 2 – 4 weeks).

3 LONG TERM CONTROL

[STEP THERAPY BASED ON ASTHMA SEVERITY]

Mild Intermittent Asthma

People with mild intermittent asthma do not need daily medication.

QUICK–RELIEVER Short Acting β2 – Agonist (SABA) **(inhaler)**, ***Salbutomol*** is used:

Only: as needed (up to a maximum of four times / day).

CONTROLLERS: long – term control medicines,

None is needed.

Mild Persistent Asthma

People with mild intermittent asthma need two types of medicines:

QUICK–RELIEVERS: Short Acting β2 – Agonist **(SABA)** **(inhaler)**, ***Salbutomol*** is used:

For immediate relief: if an asthma attack occurs.

PLUS

CONTROLLERS: long – term control medicines, [Single drug]

Inhaled corticosteroids *(low dose range ≈ 100 – 200 µg / day)* are used:

As a maintenance every day to prevent symptoms and attacks.

Moderate Persistent Asthma

People with mild intermittent asthma need two types of medicines:

QUICK–RELIEVERS: Short Acting β2 – Agonist **(SABA)** **(inhaler)**, ***Salbutomol*** is used:

For immediate relief: if an asthma attack occurs.

PLUS

CONTROLLERS: long – term control medicines, [2 drugs]

Inhaled corticosteroids *(medium dose range ≈ 200 – 400 µg / day)*,

Long Acting β2 – Agonist **(LABA)** **(inhaler)**, e.g. ***Salmetrol***

A combination of these drugs are used:

As a maintenance every day to prevent symptoms and attacks.

Severe Persistent Asthma

People with mild intermittent asthma need two types of medicines:

QUICK–RELIEVERS: short acting bronchodilator **(β2-stimulant inhaler)**, ***Salbutomol*** is used:

For immediate relief: if an asthma attack occurs.

PLUS

CONTROLLERS: long – term control medicines, [Multiple drugs]

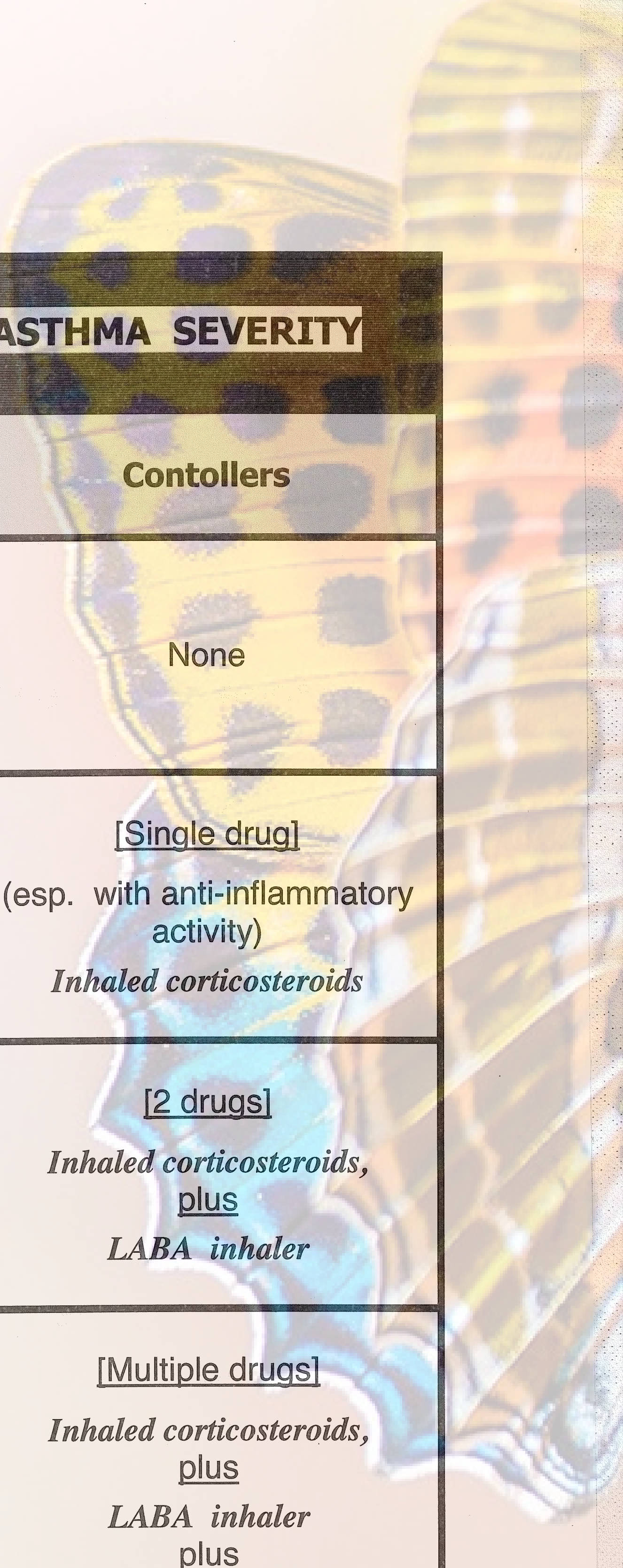
Inhaled corticosteroids *(high dose range > 400 µg / day)*,

Long Acting β2 – Agonist **(LABA)** **(inhaler)**, e.g. ***Salmetrol***

Antileukotrienes, e.g. ***Zileuton***

A combination of these drugs are used:

As a maintenance every day to prevent symptoms and attacks.



STEP THERAPY BASED ON ASTHMA SEVERITY

Classification	Quick Relievers	Contollers
STEP 1 *Mild Intermittent*	prn *	None
STEP 2 *Mild Persistent*	prn	[Single drug] (esp. with anti-inflammatory activity) *Inhaled corticosteroids*
STEP 3 *Moderate Persistent*	prn	[2 drugs] *Inhaled corticosteroids,* plus *LABA inhaler*
STEP 4 *Severe Persistent*	prn	[Multiple drugs] *Inhaled corticosteroids,* plus *LABA inhaler* plus *Antileukotrienes*

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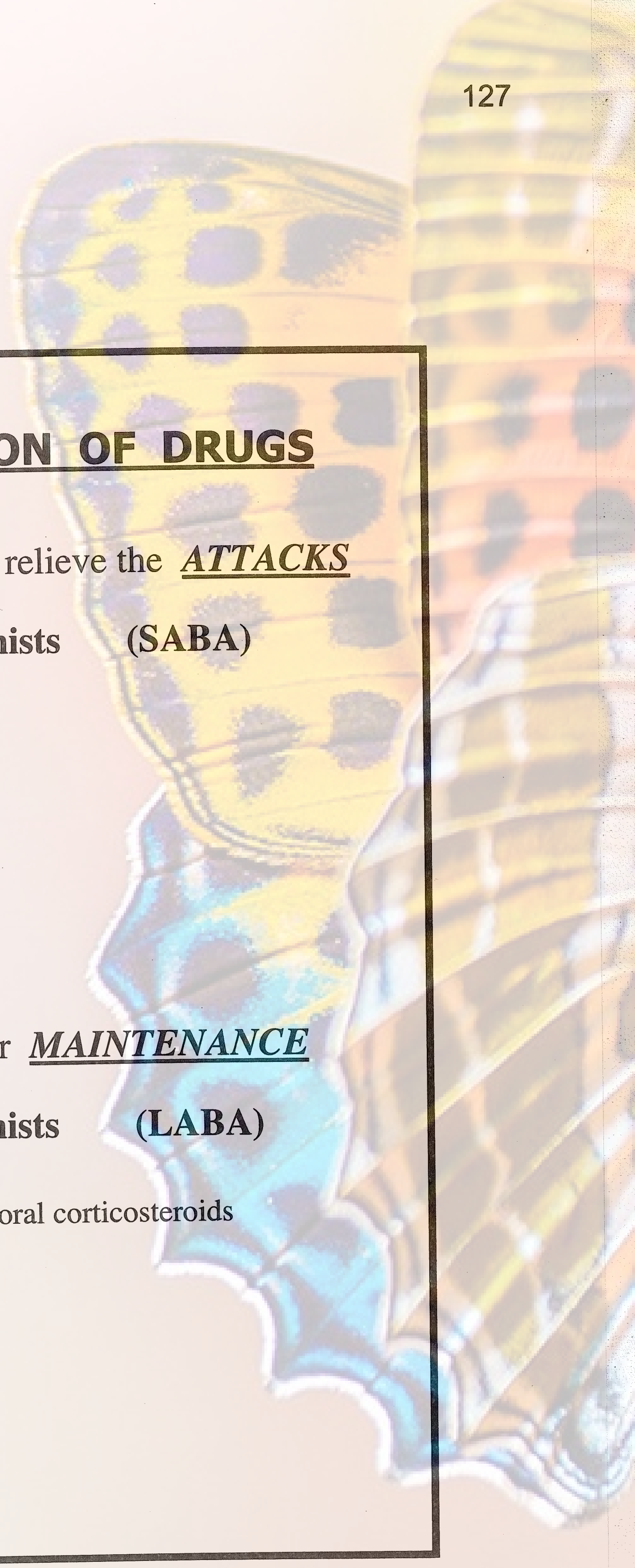
* prn = pro re nata = as needed



CLINICAL CLASSIFICATION OF DRUGS

1. **QUICK–RELIEVERS**: used to relieve the ***ATTACKS***

- **Short acting Inhaled β 2 agonists (SABA)**
- Inhaled anticholinergics
- Short acting xanthines
- IV corticosteroids

2. **CONTROLLERS**: used for ***MAINTENANCE***

- **Long acting Inhaled β 2 agonists (LABA)**
- **Inhaled corticosteroids** & oral corticosteroids
- **Antileukotrienes**
- Disodium cromoglycate
- Long acting xanthines

4 TTT OF STATUS ASTHMATICUS

A) HOSPITAIZATION: in a respiratory ICU.

B) DRUGS:

1. β_2 - Agonists: "SALBUTAMOL"
    - **IPPB:** using Nebulizer, 5 mg with O_2.

      OR
    - **Parentral:** IV infusion, 250 ug over 10 minutes,
      (If no improvement occurs with Nebulizer)
2. Inhaled anticholinergics: "IPRATROPIUM BROMIDE"
    - **IPPB:** using Nebulizer, 250 ug with nebulized salbutamol.
3. Aminophylline: (Short acting, continuous IV infusion, rarely used)
    - Loading dose: 5 mg / kg over 30 minutes.
    - Maintaine dose: 0.5 mg /kg /hour.

IF THERE IS NO RESPONSE: ADD:

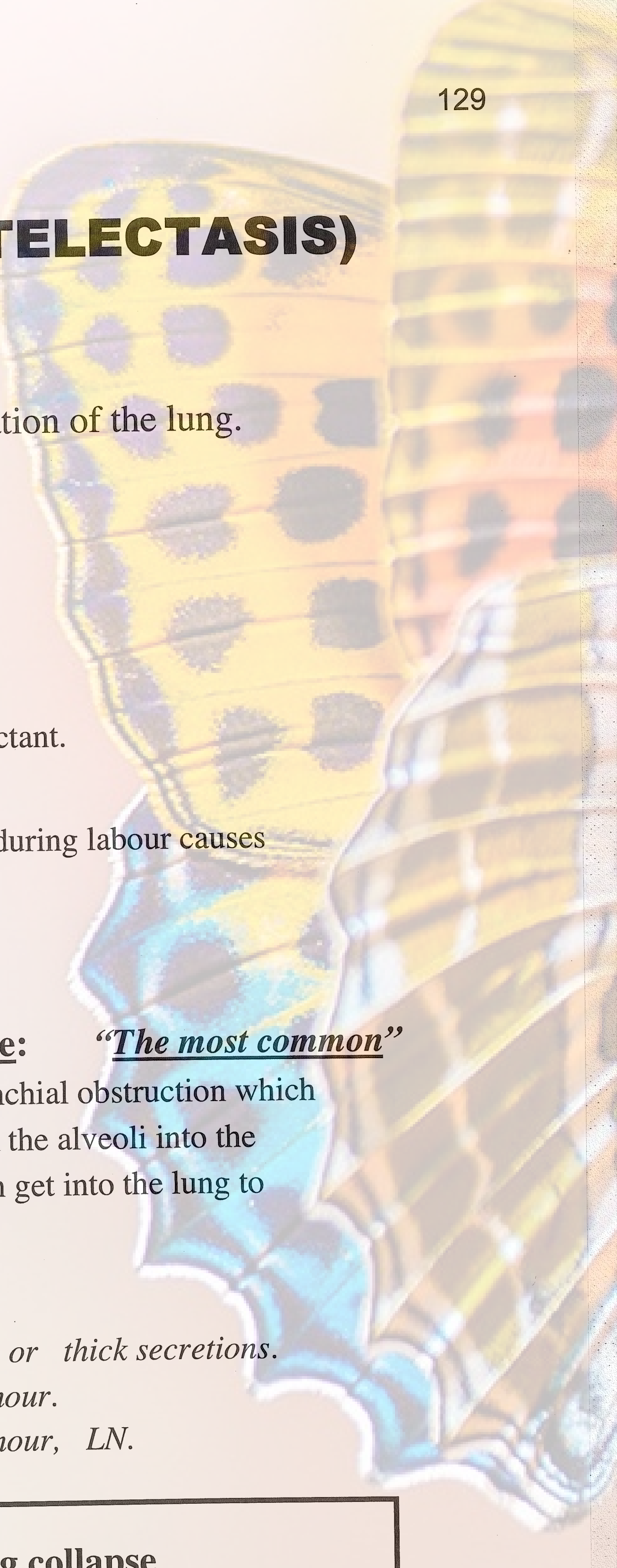
4. Systemic corticosteroids:
    - Methylprednisolone: 2 mg / Kg IV followed by 1 mg / Kg / 6 hours.
    - Hydrocortisone: 200 mg IV & repeated / 6 hours.
    - Oral prednisone: *AFTER IMPROVEMENT*
      1mg / Kg / day orally is given for 2 weeks, **then**
      the dose is gradually tappered by 5 mg every 3 days.

C) SUPPORTIVE MEASURES:

1. Oxygen inhalation.
2. Correction of: *acidosis, electrolyte imbalance.*

D) MECHANICAL VENTILATION:

- May be needed in severe resistant cases.



LUNG COLLAPSE (ATELECTASIS)

DEFINITION

- Deflation of the alveoli → reduced aeration of the lung.

ETIOLOGY

1 Neonatal collapse

1- Congenital (adhesive) collapse:

This is usually due to diminished surfactant.

2- Aspiration:

Aspiration of amniotic fluid or mucus during labour causes lack of aeration of part of the lung.

2 Acquired collapse

1- Obstructive (absorption) collapse: ***"The most common"***

- Occurs as a result of: complete bronchial obstruction which is followed by absorption of air from the alveoli into the blood, leading to collapse. No air can get into the lung to replace the absorbed air.

- Causes of bronchial obstruction:
  - In the lumen: *FB or thick secretions.*
  - In the wall: *Tumour.*
  - Pressure from outside: *Tumour, LN.*

Massive post-operative lung collapse

- A very serious example of obstructive collapse, *due to*:

1. Lack of pre-anaesthetic medication.
2. Irritation by anaesthetics especially in cases of chronic bronchitis.
3. Neglected aspiration of secretions following operations.
4. Inability to cough after operation because of pain.

2- **Relaxation-comression collapse**:

- ***In moderate cases*** of mechanical pressure, e.g. pleural effusion or pneumothorax, the intrapleural pressure rises, becomes ***less negative***, then reaches zero. This causes passive recoil of the alveoli, (**relaxation collapse**).

- ***In severe cases***, the intrapleural pressure rises more & becomes even ***positive***. This causes compression of the alveoli → more collapse, (**compression collapse**).

3- **Cicatrization collapse**:

- Severe parenchymatous fibrosis in a lung area → partial collapse.

CLINICAL PICTURE

Symptoms

1. Symptoms of the cause: **e.g.** br. carcinoma.

2. Symptoms due to collapse:

A. In most cases:

- Mild cases: *asymptomatic.*
- Severe cases: *dyspnea & may be cyanosis.*

B. In massive post-operative lung collapse: "***4 manifestations***"

- Acute chest pain.
- Dyspnea (severe) & cyanosis.
- Acute RVF.
- Shock.

Signs

1. Signs of the CAUSE: **e.g.** br. carcinoma.

2. LOCAL signs:

Inspection

- Shape: retraction on the affected side.
- Movement: decreased on the affected side.

Palpation

- **T**rachea & mediastinum: shifted to the same side.
- **T**VF: decreased on the affected side.

Percussion

- Dullness on the affected part.

Auscultation

- Breath sounds: diminished or absent.

COMPLICATIONS

1. Infection & abscess formation.
2. Bronchiectasis.
3. Failure of re-expansion after treatment.
4. Respiratory failure in massive collapse.

INVESTIGATIONS

1. CXR:

A) Features of the CAUSE: **e.g.** br. carcinoma.

B) Features of collapse:

- Opacity: taking the shape of a shrunken lobe.
- The SILHOUETTE sign.
- *The trachea & mediastinum:* ***may be shifted to the same side.***
- *The copula of the diaphragm:* ***may be elevated.***

2. Bronchoscopy:

- Diagnostic: for detection of the cause, e.g. br. carcinoma or FB.
- Therapeutic: for removal of the obstructing material, *if possible*.

DIFFERENTIAL DIAGNOSIS

1. Causes of: **" syndrome of multiple negatives"**
   Pleural effusion, lung collapse, lung fibrosis.
2. Consolidation.
3. Differentiation of: **"acute massive post-operative lung collapse"**
   Other causes of: ***acute chest pain***, ***dyspnea***, ***acute RVF*** & ***Shock***:
   - Massive myocardial infarction.
   - Massive pulmonary embolism.
   - Tension pneumothorax.

TREATMENT

1. Treatment of the cause: e.g.

A) Obstructive (absorption) collapse:

- Bronchoscopic removal of foreign bodies or secretions.
- The patient is placed on the healthy side & the affected side is heavily percussed to dislodge the obstructing material.

B) Relaxation-comression collapse:

- TTT of the cause: e.g. pleural effusion or pneumothorax.

2. Treatment of complications: e.g.

- Antibiotics for infection.

3. Oxygen inhalation.

4. Respiratory exercises.

PULMONARY FIBROSIS

TYPES

1 Pulmonary fibrosis

I. *Parenchymatous fibrosis*:

- Occurs as a complication of various lung diseases, **e.g.**
    - TB.
    - Lung abscess.
    - Bronchiectasis.

II. *Interstitial fibrosis*: (ILD)

- Occurs in between the alveoli, in the interstitial tissue.

2 Pleuro-Pulmonary fibrosis " fibrothorax "

- Pleural diseases may heal by extensive fibrosis that extends to the lung parenchyma.
- It commonly complicates:
    - Hemothorax.
    - Empyema.

Parenchymatous fibrosis & Fibrothorax

CLINICAL PICTURE

Symptoms

1. Symptoms of the cause: **e.g. TB toxemia**.
2. Symptoms due to fibrosis:
    - Dyspnea.
    - Dry cough.
    - Chest pain (in fibrothorax).

Signs

1. Signs of the CAUSE: **e.g. TB**.
2. LOCAL signs:

Inspection

- Shape: retraction on the affected side.
- Movement: diminished on the affected side.

Palpation

- **T**rachea & mediastinum: shifted to the same side.
- **T**VF: decreased.

Percussion

- Dullness: patchy (heterogenous).

Auscultation

- Breath sounds: diminished.
- Additional sounds: crepitations.

COMPLICATIONS

1. Bronchiectasis.
2. Corpulmonale.
3. Cicatrization collapse.
4. Respiratory failure: *in severe bilateral cases*.

INVESTIGATIONS

1. CXR:

A) Features of the CAUSE: **e.g.** TB.

B) Features of fibrosis:

- Opacity: heterogenous with reticulations.
- *The trachea & mediastinum:* ***may be shifted to the same side***.
- *The copula of the diaphragm:* ***may be elevated*** (TENTING).

2. Respiratory function tests:

- Evidence of: RESTRICTIVE HYPOVENTILATION.

DIFFERENTIAL DIAGNOSIS

- Causes of: **" syndrome of multiple negatives"**

  Pleural effusion, lung collapse, lung fibrosis.

TREATMENT

- Treatment of the cause, e.g. TB.
- Treatment of the complications, e.g. Respiratory failure.

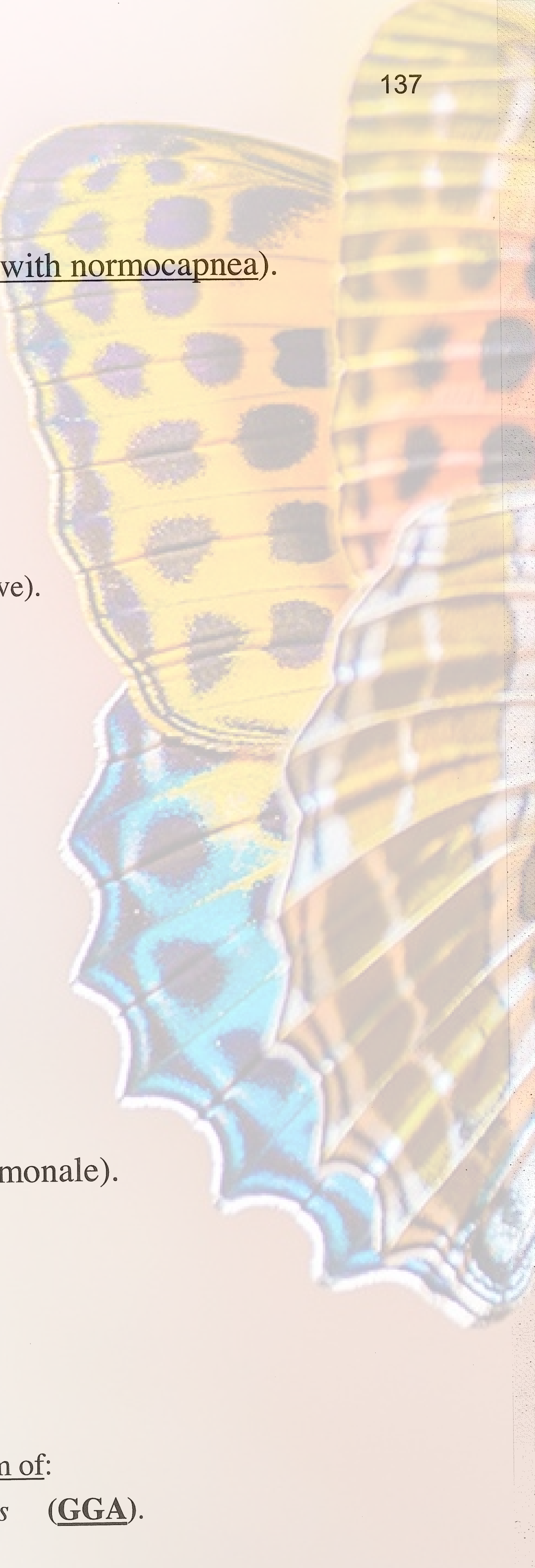
Interstitial Lung Fibrosis (ILD)

DEFINITION

- Diffuse inflammatory disorders of the lower respiratory tract characterized by: ***alveolitis***, ***thickening of alveolar walls***, ***extensive fibrosis*** of the interstitial tissue.

ETIOLOGY

1. **Idiopathic pulmonary fibrosis** **(IPF).**
2. **Immune diseases:**
   - Collagen diseases.
   - Good Pasture's syndrome.
3. **Inflammatory bowel diseases:**
   - Crohn's disease.
   - Ulcerative colitis.
4. **Inhalational lung diseases:**
   - Extrinsic allergic alveolitis ***(due to organic dust)***
     - Farmer's lung.
   - Pneumoconiosis ***(due to inorganic dust)***
     - Silicosis.
     - Asbestosis.
5. **Iatrogenic:**
   - Amiodarone.
6. **Hepatic diseases:**
   - Chronic active hepatitis.
   - Primary biliary cirrhosis.
7. **Heart diseases:**
   - Chronic LSHF.
8. **SARCOIDOSIS.**



PATHOPHYSIOLOGY

- IMPAIRED DIFFUSION (Hypoxia with normocapnea).
- Restrictive hypoventilation.

CLINICAL PICTURE

Symptoms

- **D**yspnea (gradual & progressive).
- **D**ry cough.

Signs

- Central cyanosis.
- Clubbing.
- Crepitations (diffuse).

COMPLICATIONS

1. **R**espiratory failure.
2. **R**ight ventricular failure (corpulmonale).

INVESTIGATIONS

1. CXR:

- Bilateral diffuse lung affection in the form of: *reticulations & micronodular infiltrations* (**GGA**).
- Pulmonary hypertension ± right ventricular enlargement.

2. Pulmonary function tests:

- *Ventilation tests:*
  Restrictive hypoventilation.
- *Diffusion tests:*
  Decreased CO transfer factor.

3. Arterial blood gases:

- Hypoxia.
- Normocapnea.

4. Assessment of disease activity: (active alveolitis)

- ***BAL:*** for detection of type & amount of inflammatory cells in active alveolitis.
- ***Gallium-67 scan:***
  - The affected lung tissue (with active alveolitis) takes up Gallium-67.
  - The normal lung tissue does not take up Gallium-67.
- ***Open lung biopsy:*** for detection of active alveolitis.

5. Investigations for the cause.

TREATMENT

1. Treatment of the cause.
2. Treatment of ***Idiopathic Pulmonary Fibrosis***:
   - Corticosteroids: prednisone: 1 mg / kg / day.
   - Cytotoxic drugs: **e.g.** cyclophosphamide.
3. Symptomatic treatment:
   - Oxygen therapy.
4. Treatment of complications:
   - Respiratory failure.
   - Right ventricular failure.

COR PULMONALE

DEFINITION

Right ventricular hypertrophy with or without failure, resulting from PH, due to: _parenchymal_ or _vascular_ lung disease.

ETIOLOGY

A) Acute cor pulmonale:

1. Massive pulmonary embolism.
2. Tension pneumothorax.
3. Massive lung collapse.

B) Subacute cor pulmonale:

1. Lymphagitis carcinomatosis.
2. Recurrent showers of pulmonary emboli.

C) Chronic cor pulmonale:

1. Hypoxic cor pulmonale:

   Occurs in almost all cases of chronic lung diseases, **e.g.**

   - Obstructive hypoventilation **e.g.** COPD.
   - Restrictive hypoventilation **e.g.** ILD.

2. Vascular cor pulmonale:

   - Primary pulmonary hypertension.
   - Pulmonary bilharziasis.
   - Pulmonary vasculitis.
   - Sickle cell anemia.
   - Recurrent showers of pulmonary emboli.

CLINICAL PICTURE

1- Features of: the cause.
2- Features of: pulmonary hypertension.
3- Features of: right ventricular enlargement.
4- May be features of: right ventricular failure.

INVESTIGATIONS

1. **CXR**:
    - Features of: the cause.
    - Features of: pulmonary hypertension.
    - RV enlargement.
    - RA enlargement.

2. **ECG**:
    - RV enlargement.
    - RA enlargement (p – pulmonale).

3. **Echocardiography**:
    - RV enlargement.
    - RA enlargement.

TREATMENT

1- Treatment of the cause.

2- Treatment of pulmonary hypertension: (Refer to Cardiology).

3- Treatment of right-sided heart failure: (Refer to Cardiology).

HYPOXIC VERSUS VASCULAR COR PULMONALE

	Hypoxic cor pulmonale	Vascular cor pulmonale
Dyspnea	Marked	Minimal or absent
Suptum	Mucopurulent	Absent
Cyanosis	Central due to hypoxia	Peripheral due to low CO
Clubbing	Common	Absent
Chest Examination		
• *Shape*	Barrel –shaped	Normal
• *Breath sounds*	Prolonged expiration	Normal
• *Additional sounds*	Wheezes	Absent
• *Features of PH*	May be masked	Marked
ABG	Hypoxia, may be hypercapnia	Usually normal

Bilharzial Cor Pulmonale

ETIOLOGY

1. Schistosoma mansoni ova:

- They reach the lungs only in the presence of portal hypertension and porto-systemic shunts which allow the ova to bypass the liver to the lungs.
- They cause ***more destructive lesions*** than S. haematobium.

2. Schistosoma haematobium ova:

- They reach the lungs more commonly since the worms live in the pelvic venous plexuses which drain into the IVC, then to the lungs.
- They usually produce ***mild lesions***.

PATHOLOGY

1. Pulmonary hypertension:

- The bilharzial ova reach the lungs as emboli leading to necrotising arteriolitis due to the secretion of miracidial toxins. This is followed by: ***end-arteritis obliterans*** with obliteration of pulmonary arterioles leading to: pulmonary hypertension.
- ***Bilharzial immune complexes*** may play a role through causing pulmonary vasculitis, leading to pulmonary hypertension.

2. Cor pulmonale:

- Pulmonary hypertension → RVH & eventually RVF (cor pulmonale).

3. Aneurysm formation:

- Aneurysmal dilatation of the pulmonary artery & its two main branches occur due to progressive weakness in the wall of the arteries.

CLINICAL PICTURE

Symptoms

- Symptoms of: low CO.
- Symptoms of: RVF may occur lately.
- No central cyanosis.

Signs

- Signs of: pulmonary hypertension.
- Signs of: right ventricular enlargement.
- Signs of: RVF may occur lately.
- Signs of: PULMONARY ANEURYSM:

  "marked pulsations & dullness in the left second left space".
- Hepatosplenomegaly & signs of portal hypertension.

INVESTIGATIONS

1. CXR:

- Marked dilatation of the main pulmonary arteries forming:

  " *Dumb-bell shaped shadows in the hila* ".
- Peripheral pulmonary oligemia.
- RV enlargement.
- RA enlargement.

2. ECG:

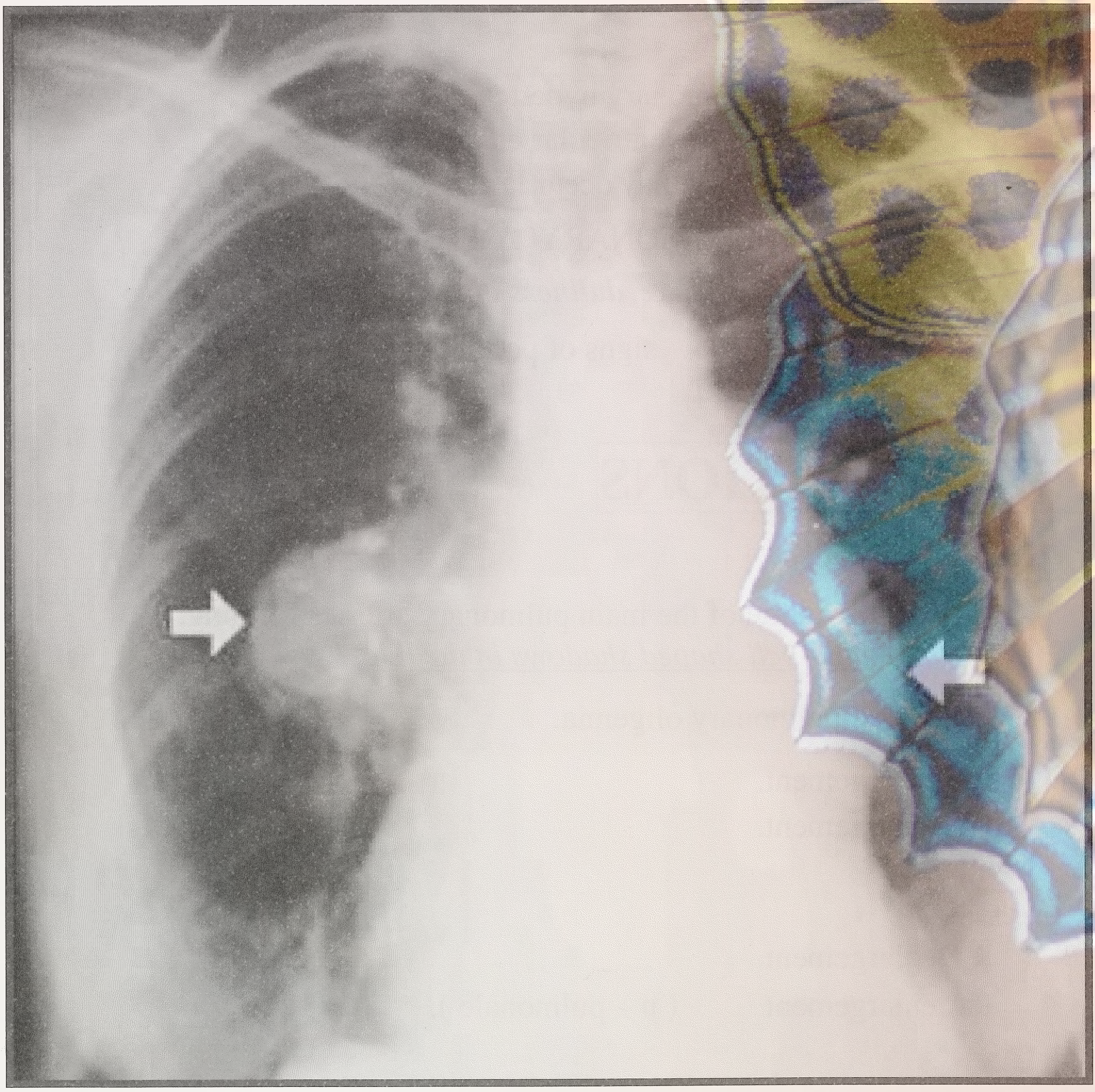
- RV enlargement.
- RA enlargement (p – pulmonale).

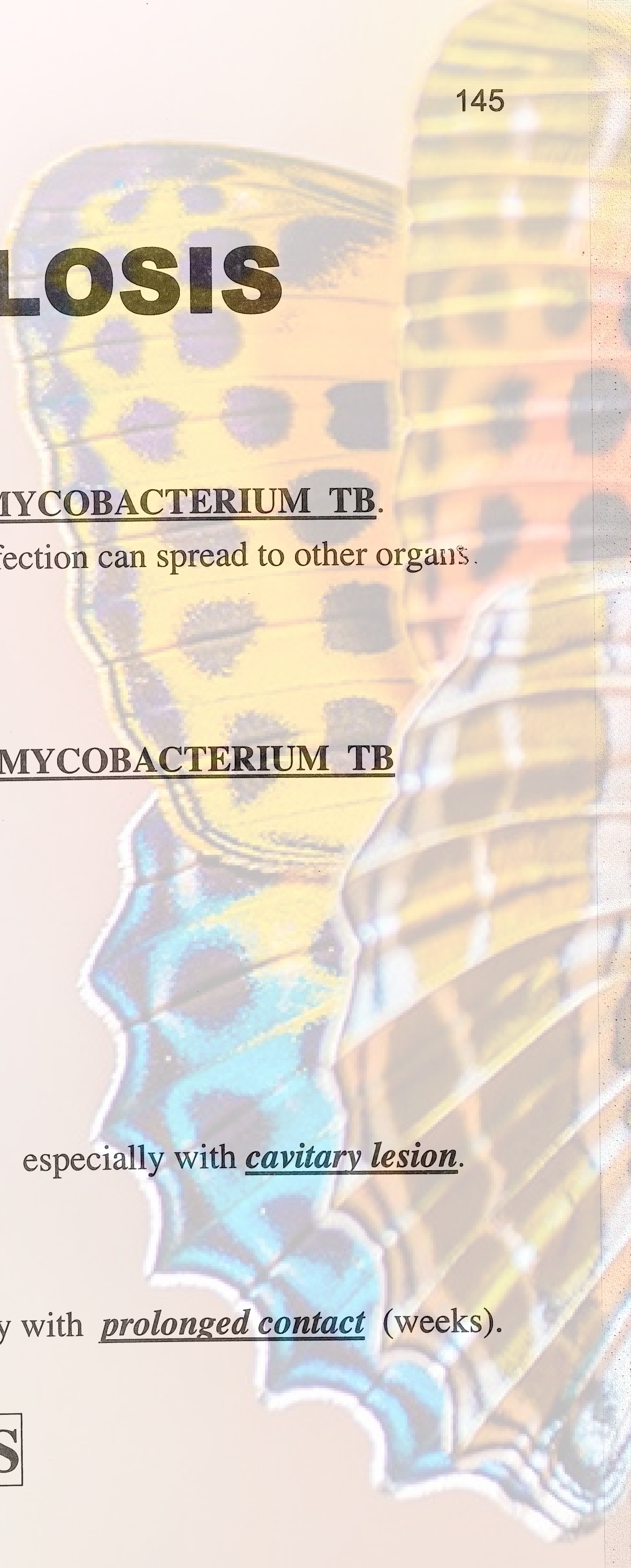
3. Investigations for bilharziasis: (refer to GIT).

TREATMENT

1) Proper treatment of cor pulmonale.
2) Anti-bilharzial treatment.

Marked dilatation of the main pulmonary arteries forming:
" *Dumb-bell shaped shadows in the hila* "





TUBERCULOSIS

DEFINITION

- It is a common contagious infection caused by **MYCOBACTERIUM TB**.
- It primarily affects the **LUNGS**, however: infection can spread to other organs.

ETIOLOGY

1. **Causative organism**: **MYCOBACTERIUM TB**
    - **A**cid – fast.
    - **A**lcohol – fast.
    - **A**erobic.
2. **Source of infection**:
    - Patients with **A**ctive pulmonary TB, especially with ***cavitary lesion***.
3. **Mode of infection**:
    - **A**irway Droplet infection, especially with ***prolonged contact*** (weeks).

PREDISPOSING FACTORS

1. Age:
    - Below 5 y: High succeptibility to infection.
    - 5 – 15 y: Less susceptibility (***golden age***).
    - Above 15 y: High susceptibility to progressive pulmonary disease.
2. Race: more common in black races.
3. Incidence: "Poor countries & Poor socio-economic classes"
    - **P**oor nutrition, Poor hygienic conditions.

4. Occupation:
    - Paramedical & medical people are more exposed to infection.
5. Chest diseases:
    - Pneumoconiosis: e.g. Silicosis, asbestosis ↓ resistance to infection.
    - Pulmonary oligaemia: e.g. PS.
6. Decreased immunity:
    - **D**iabetes mellitus.
    - **D**rugs: *Corticosteroids* & *other immunosuppressives*.
    - **A**IDS.
    - **M**alignancies, *especially lymphomas*.
    - **M**alnutrition.

PATHOGENESIS

I. ENTRY

Inhaled TB bacilli travel via the airways to be deposited in the pulmonary parenchyma, especially in the ***upper zones***, probably due to high O_2 tension which favours the growth of the organisms.

II. FIGHT

Alveolar macrophages ingest TB bacilli & try to phagocytize them.

Alveolar macrophages usually fail to completely destroy the bacilli which possess natural defences. They are usually ***not destroyed***.

There is a fight between the: Alveolar macrophages & TB bacilli.

III. FATE

1. Primary TB: (Childhood)

a) Latent form:

In most immunocompetent individuals, macrophages are successful in inactivating the bacilli. The bacilli stay: LATENT or DORMANT.

Therefore the infection is self-limited and often ***subclinical***.

Patients are asymptomatic: and the underlying lung pathology is:

The primary complex: Gohn's focus, regional lymphadenitis, lymphangitis

b) Active (Progressive) form:

In some patients, macrophages fail to inactivate the bacilli.

The bacilli become active leading to a ***clinically apparent*** infection.

Patients may present with: pulmonary or extrapulmonary manifestations

2. Postprimary (reactivation) TB: (Adulthood)

After a period of latent primary TB, reactivation of the disease may occur at any time if the immunity of the patient drops.

Patients may present with: pulmonary or extrapulmonary manifestations

PATHOLOGY

1. Primary TB: (Childhood)

a) Gohn's focus: (pulmonary component)

An area of consolidation formed by aggregation of tubercles, present usually in the mid to upper zones, usually supleural.

b) Regional lymphadenitis (glandular component)

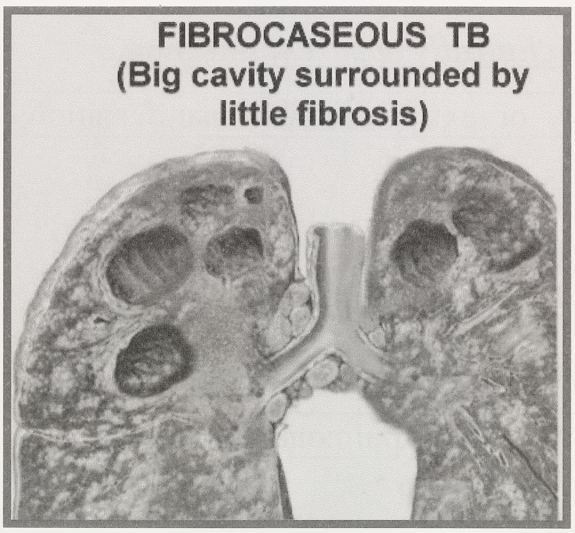
c) Lymphangitis in the intervening lymphatic vessels.

2. Postprimary (reactivation) TB: (Adulthood)

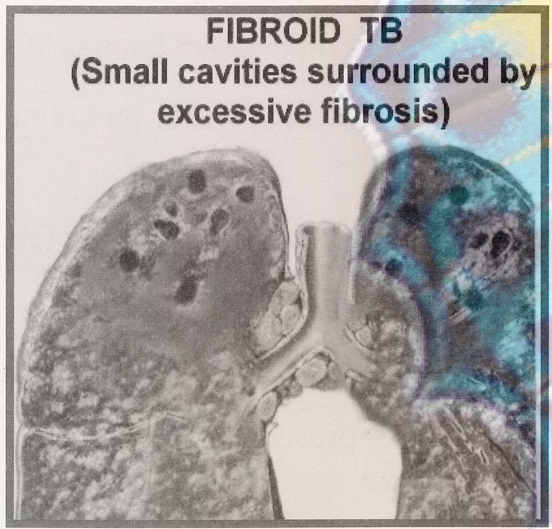
a) Gross:

- This depends on the *state of immunity* & the *virulence of the organism*:

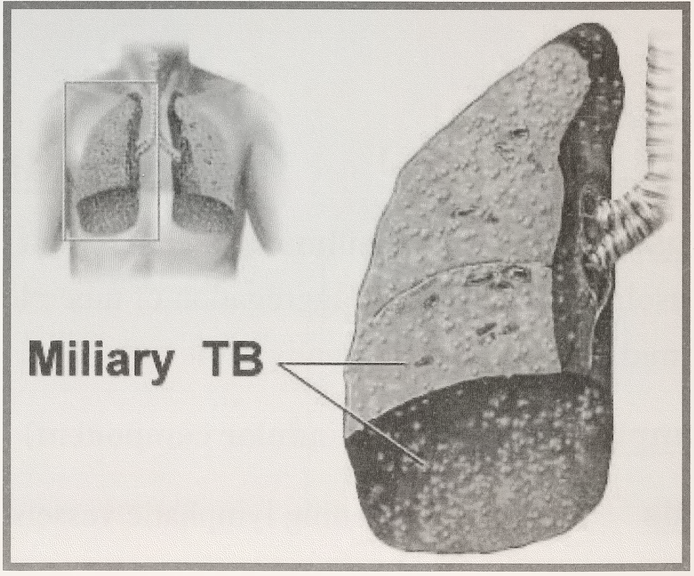
1. FIBROCASEOUS TB:
   - Big **cavity** surrounded by little fibrosis.
2. FIBROID TB:
   - Small cavities surrounded by excessive **fibrosis**.
3. Endobronchial TB:
   - TB bronchitis of the bronchi connected to TB cavities.
4. TB bronchopneumonia & Miliary TB:
   - These types occur in immunocompromised patients.



FIBROCASEOUS TB
(Big cavity surrounded by little fibrosis)

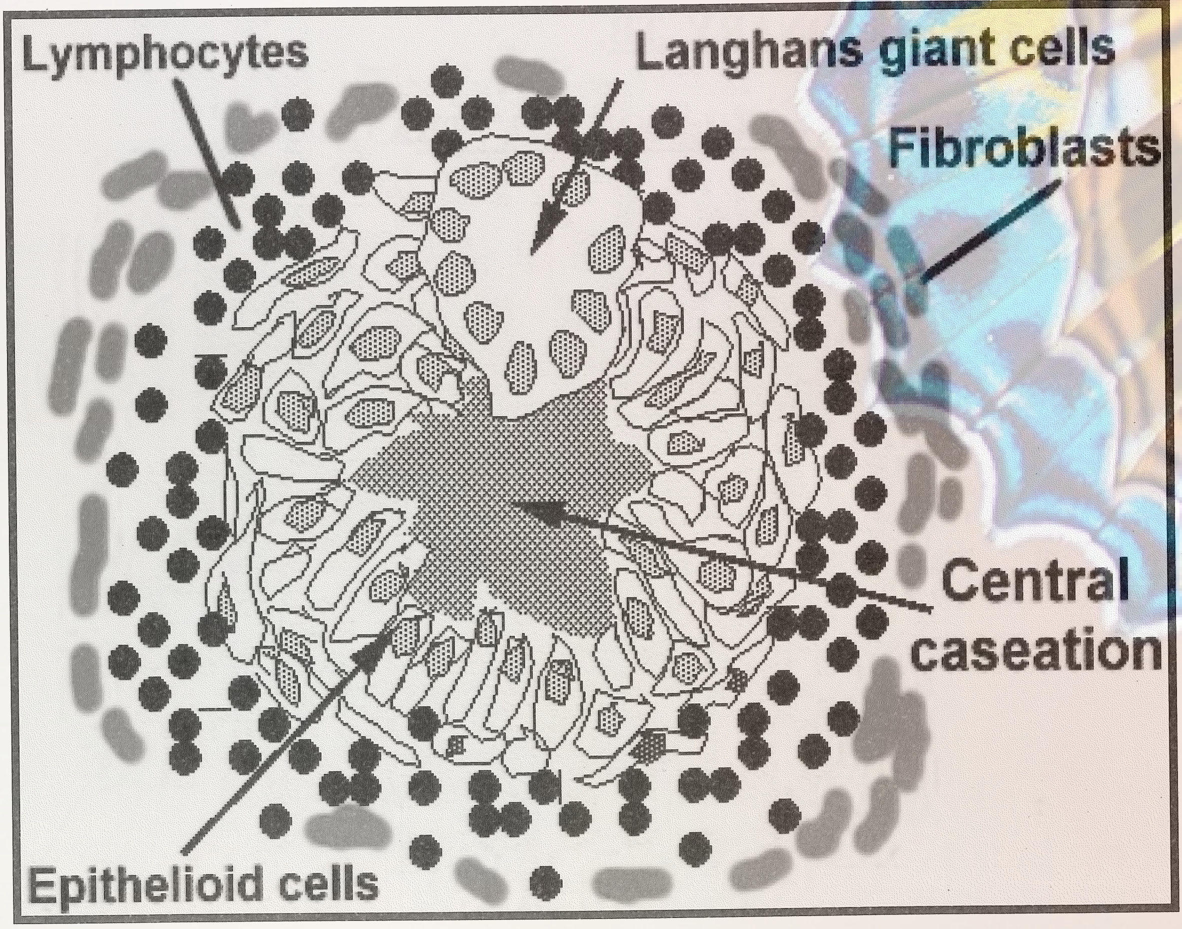


FIBROID TB
(Small cavities surrounded by excessive fibrosis)

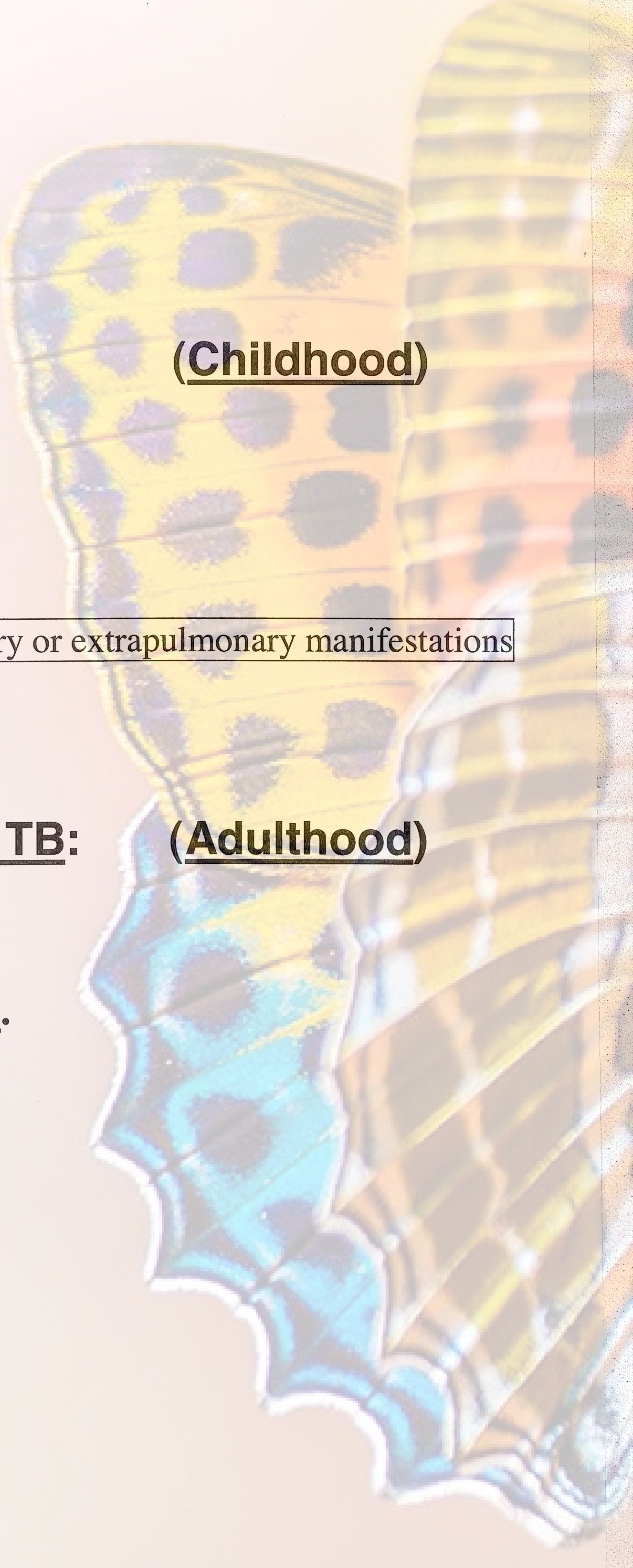


b) Microscopic:

- The characteristic lesion is: " **Typical TB granuloma** "
  - CASEATION: central, surrounded by:
  - CELLS:
    - *Lymphocytes.*
    - *Langhans giant cells.*
    - *Epithelioid cells.*
    - *Fibroblasts.*
  - CALCIFICATION.


Lymphocytes
Langhans giant cells
Fibroblasts
Central caseation
Epithelioid cells

Typical TB granuloma



CLINICAL TYPES

1. **Primary TB**: (**Childhood**)

a) **Latent form**:

Patients are usually asymptomatic.

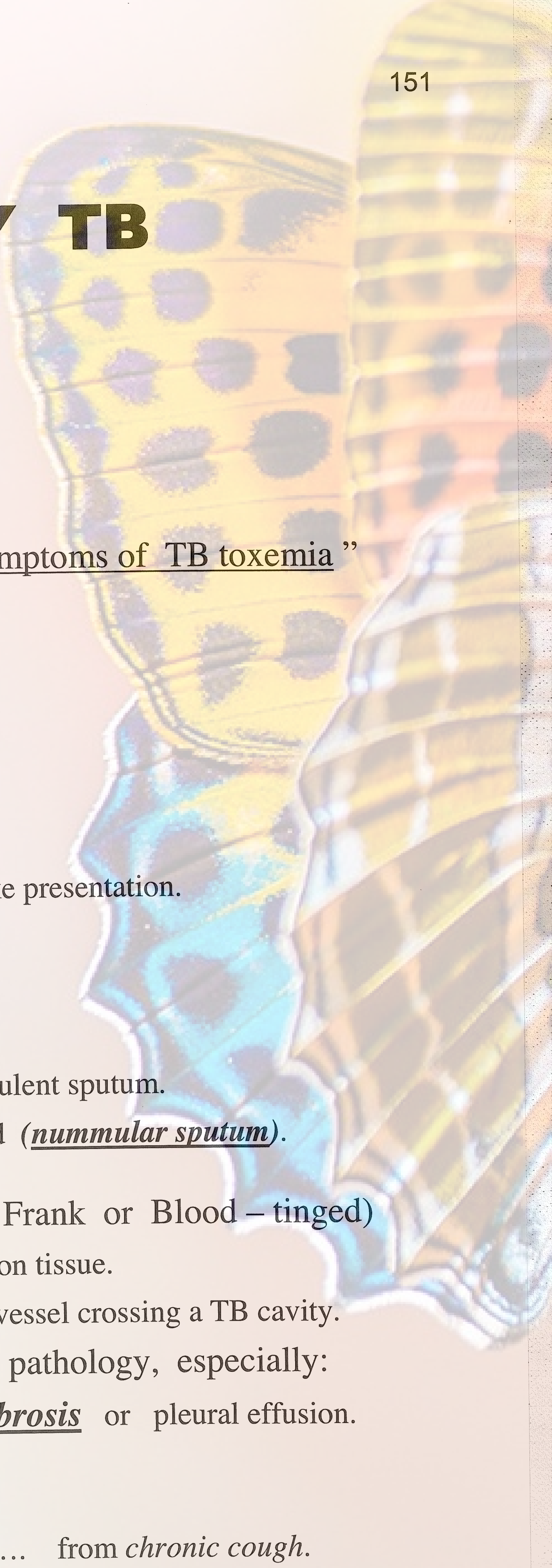
b) **Active (Progressive) form**:

Patients may present with: pulmonary or extrapulmonary manifestations

2. **Postprimary (reactivation) TB**: (**Adulthood**)

a) **PULMONARY TB**.

b) **EXTRA – PULMONARY TB**.



PULMONARY TB

CLINICAL PICTURE

Symptoms

1. **General symptoms:**

   a) *Chronic presentation:* " Symptoms of TB toxemia "

   - **Night** sweat.
   - **Night** fever.
   - ***Loss*** of weight.
   - ***Loss*** of appetite.

   b) *Acute presentation:*

   - Flu-like ***or*** Typhoid-like presentation.

2. **Chest symptoms:**

   a) *Cough:*

   - With expectoration of a mucopurulent sputum.
   - Which may be rarely coin-shaped (***nummular sputum***).

   b) *Hemoptysis:* (Common: Frank or Blood – tinged)

   - Bleeding from vascular granulation tissue.
   - Bleeding due to erosion of a big vessel crossing a TB cavity.

   c) *Dyspnea:* due to any lung pathology, especially:

   - Consolidation, ***cavitation, fibrosis*** or pleural effusion.

   d) *Chest pain:* due to:

   - Pain in the intercostals muscles …. from *chronic cough.*
   - Pneumothorax.
   - Pleurisy.

Signs

1. **General signs:**
   - Fever, toxic facies, loss of weight.
   - Clubbing: in advanced untreated cases with severe toxemia.
   - PALLOR.

2. **Chest signs:** (**variable depending on the lesion**)
   - Apical: ***cavitaion,*** ***fibrosis***. (the most common)
   - Others: consolidation,
     bronchopneumonia, bronchiectasis,
     pleural effusion, pneumothorax.

COMPLICATIONS

1. **AMYLOIDOSIS** (Secondary).
2. **Aspergilloma** (formed in a TB cavity).
3. **SPREAD** (Extra – pulmonary TB).
4. **COMPLICATIONS OF ANTI – TB drugs**.

INVESTIGATIONS

I. LABORATORY INVESTIGATIONS

1. General: (evidence of tissue inflammation)

a) ESR: markedly elevated.

b) CRP: elevated.

c) CBC:

- *WBCs: lymphocytosis & monocytosis.*
- *RBCs: anemia.*

2. Bacteriology:

a) Sputum: for TB bacilli

- *Staining: ZN stain.*
- *Culture: Lowenstein-Jensen medium or BACTEC.*
- *Animal Innoculation: Guinae pig.*

b) BAL: for TB bacilli (if no sputum is available).

3. Tuberculin test: (Mantoux test)

a) Method:

- Injection of 0.1 cc of 1/10,000 PPD (Purified Protein Derivative). *ID in the upper part of the front of the forearm.*
- The result is recorded after 72 hours.
- **Positive tuberculin:** indurated area of 1 cm or more in diameter.

b) Values:

1. **Positive** test indicates:
   - Infection with TB (whether *active* or *old*).
2. **Positive** test in children below 3 years usually indicates:
   - *Active* infection.
3. **Negative** test usually indicates:
   - Absence of infection with TB.
4. **Negative** test in adults susceptible to infection ***(Med, Paramed)*** indicates:
   - The need for BCG vaccination.

c) False positive tuberculin test:

1. Atypical mycobacteria.
2. Nocardia.
3. PREVIOUS BCG VACCINATION.

d) False negative tuberculin test:

1. Fulminant cases, e.g. acute miliary TB.
2. Decreased immunity: Lymphoma, AIDS, Corticosteroids.
3. Inactive tuberculin.

4. PCR: *(Polymerase Chain Reaction)*

- For detection of: ***Mycobacterial DNA***.

II. IMAGING

1. CXR:

- Radiologic findings are more prominent in the upper lung zones.
- Radiologic findings are variable depending on the lesion:

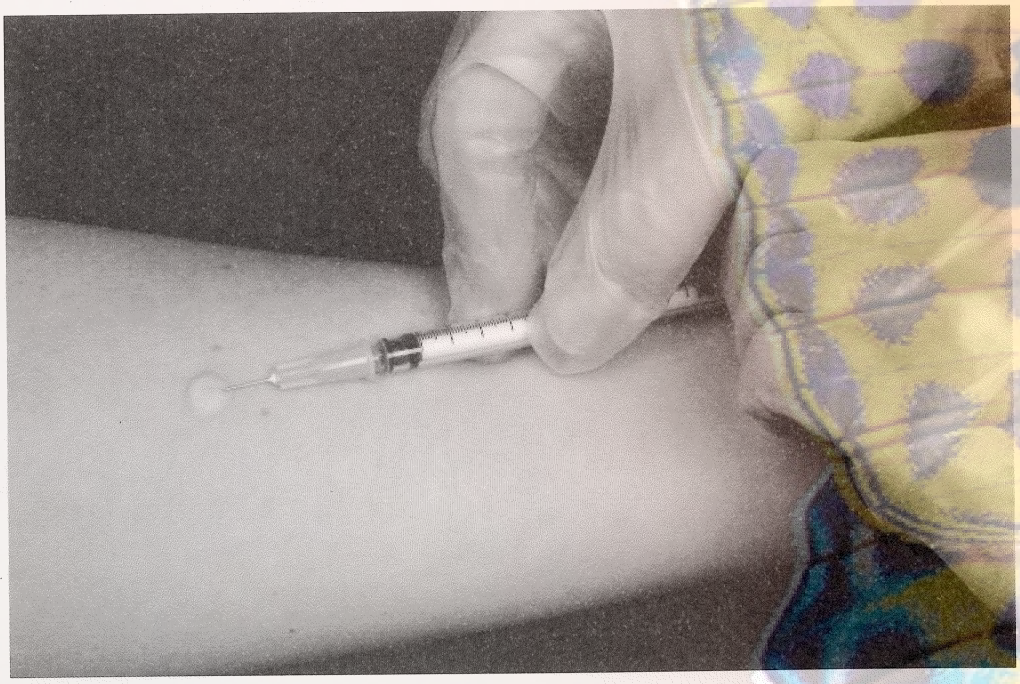
1. Fibrocaseous TB: apical cavities surrounded by opacities.
2. TB bronchopneumonia: irregular patches scattered in the lungs.
3. Miliary TB: numerous small shadows of equal sizes (GGA).
4. Others:
    - *Fibrosis.*
    - *Calcification.*
    - *Enlarged hilar LN.*
    - *Pleural effusion.*

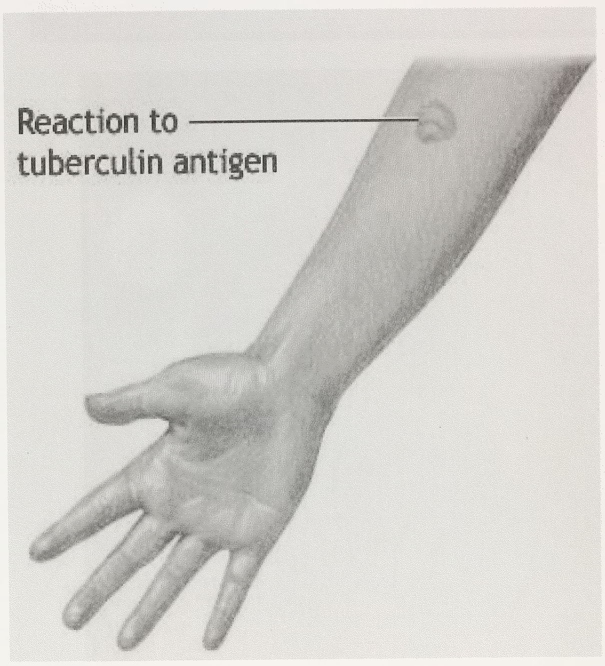
2. CT scan.

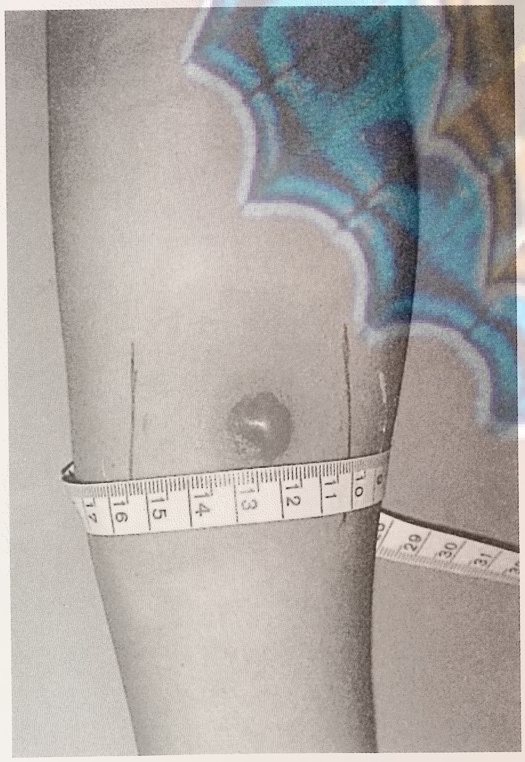
III. BIOPSIES (Transbronchial, Pleura, LN):

- " **Typical TB granuloma** " (see before).

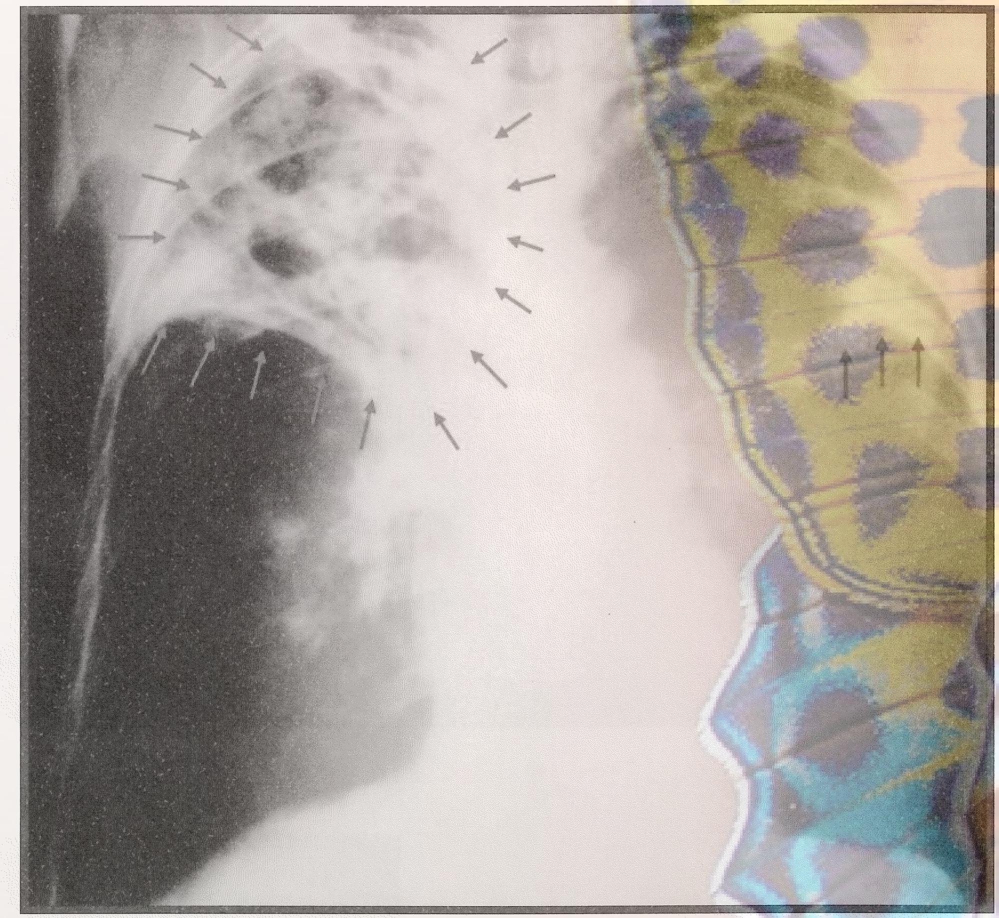
Tuberculin test

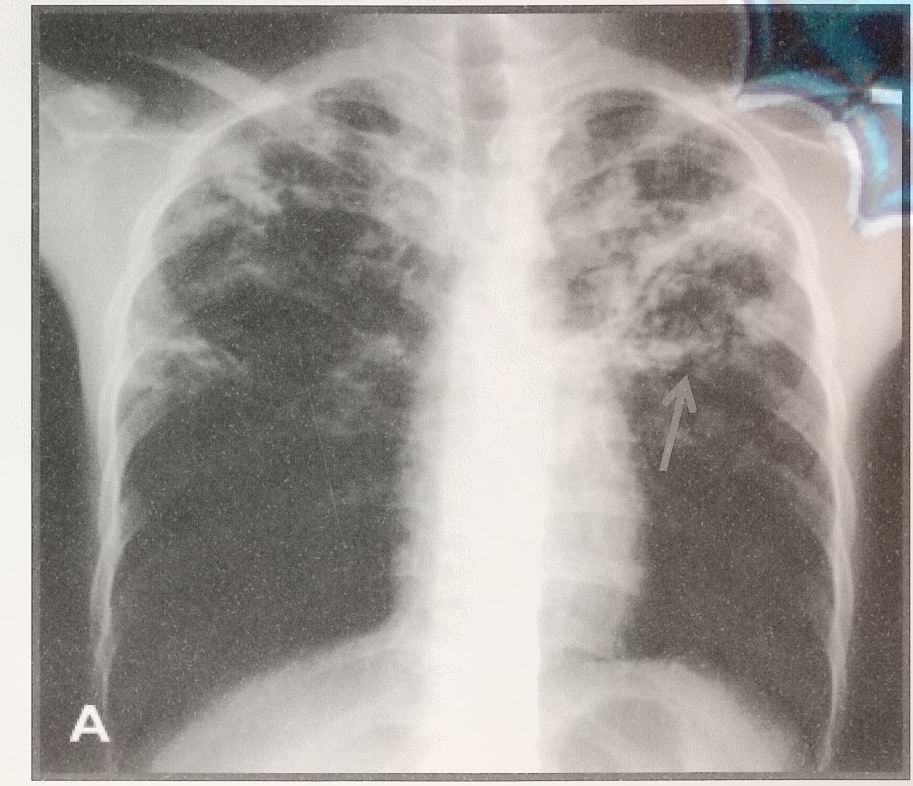



Reaction to
tuberculin antigen

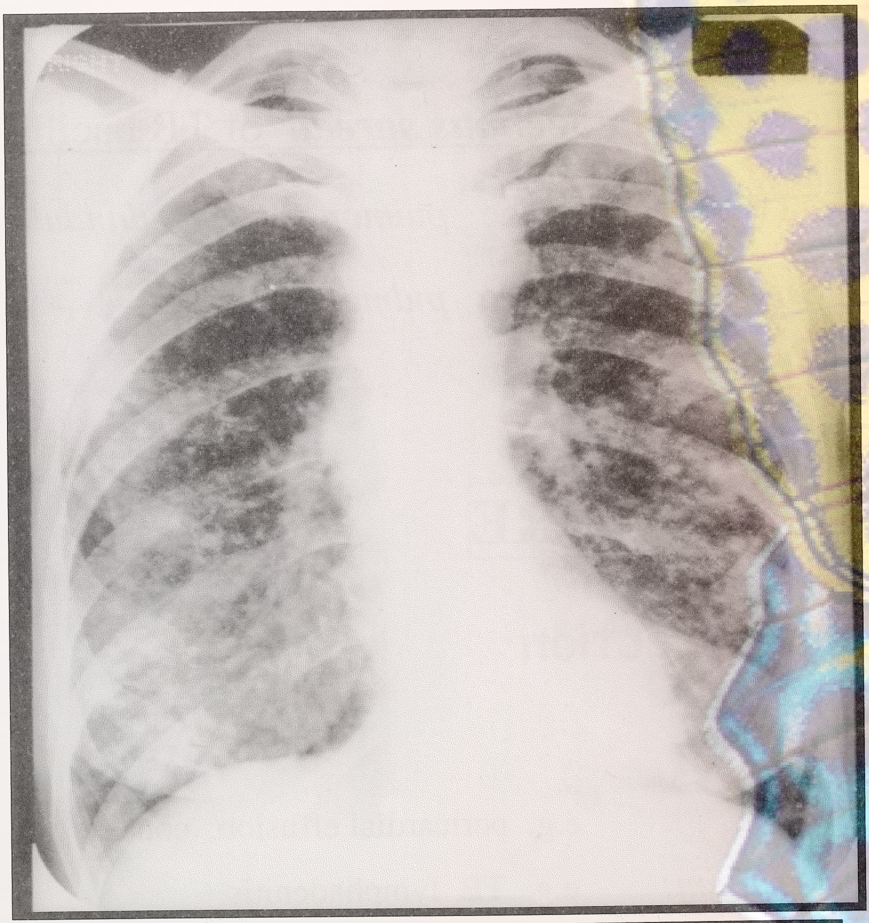


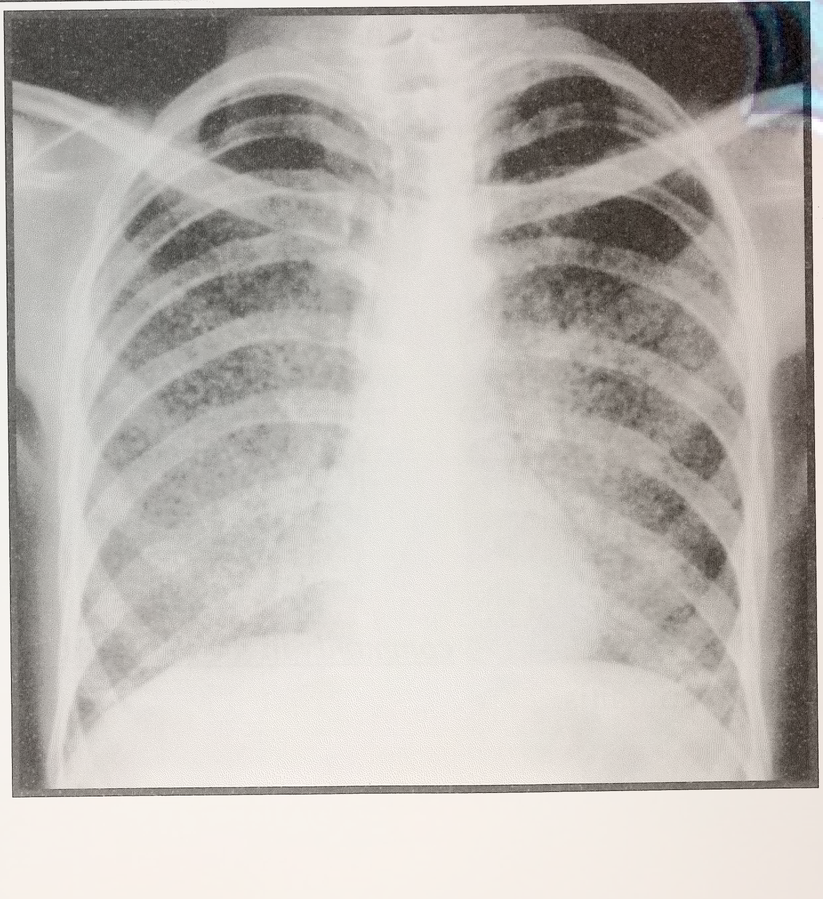
Apical cavitation surrounded by some fibrosis





Miliary TB (GGA)





EXTRA – PULMONARY TB

- It occurs due to ***Hematogenous spread*** of TB bacilli from:
    1. Primary TB: from the *pulmonary* *or* *glandular component.*
    2. Postprimary TB: from the *pulmonary lesion.*

CLINICAL PICTURE

Single organ affection

1. **Thoracic:**
    - a) *Heart:* e.g. pericardial effusion, constrictive pericarditis.
    - b) *Mediastinum:* e.g. TB lymphadenitis.
    - c) *Chest wall:* e.g. ribs (osteomyelitis).

2. **Extra - Thoracic:**
    - a) *Lymphatic:*
        - TB lymphadenitis.
    - b) *CNS:*
        - TB meningitis.
        - Tuberculoma.
    - c) *Abdominal:*
        - TB enteritis: *commonly in the **ileocecal region***.
        - TB peritonitis.

d) *Genitourinary:*

- Urinary: *Kidneys, ureter, UB.*
- Male genital: *Prostate, epididymis, seminal vesicles.*
- Female genital: *Uterus, ovaries, Fallopian tubes.*

e) *Skeletal:*

- TB osteomyelitis.
- TB orthritis.
- POTT'S DISEASE OF THE SPINE.

f) *OTHERS:*

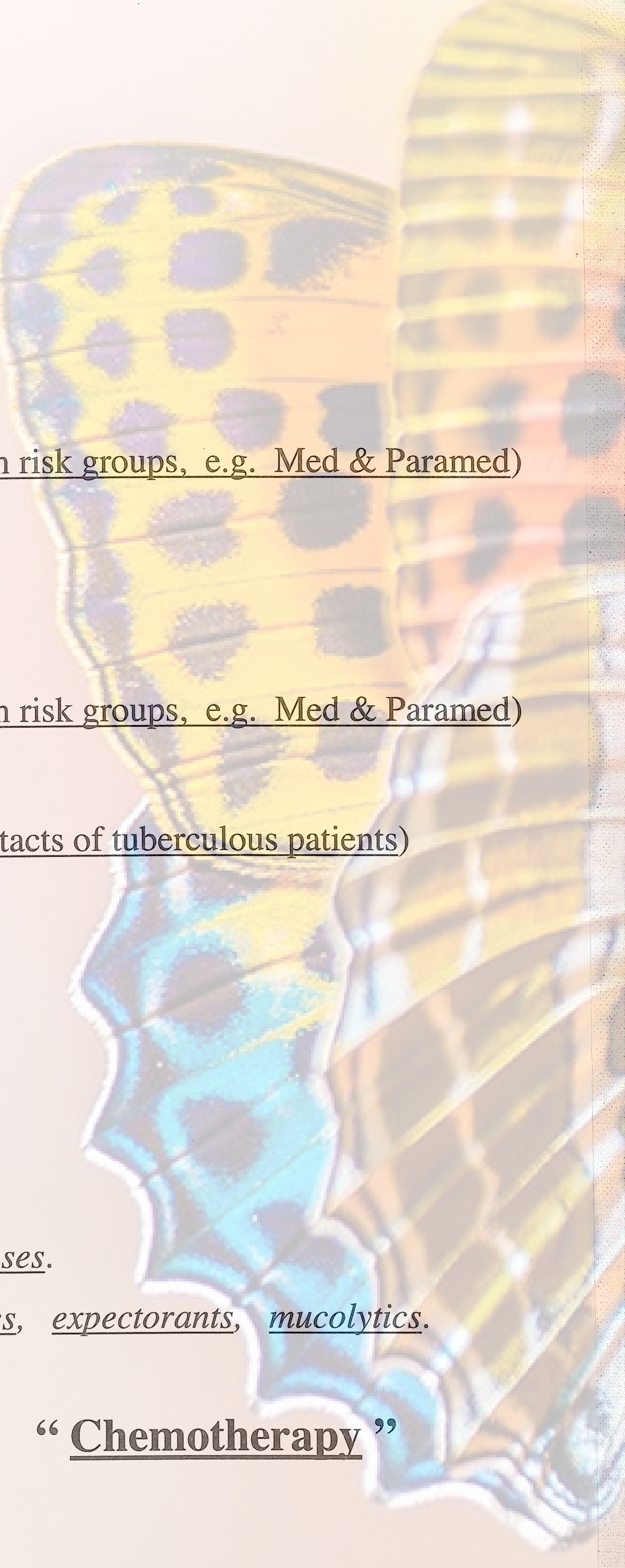
- Adrenals: Addison's disease.
- Skin: Lupus vulgaris.

Miliary TB

1. **General systemic manifestations:**
    - Fever, CYANOSIS, toxic facies, loss of weight, anorexia, sweats.
2. **Chest manifestations:**
    - Cough, dyspnea, generalized crepitations.
3. **Other manifestations:**
    - HSM, Meningitis, Polyserositis, EYE (choroid tubercles).

INVESTIGATIONS OF MILIARY TB

1. Tuberculin test: may be false negative.
2. CXR: numerous small shadows of equal sizes (**GGA**).
3. BIOPSY: transbronchial, liver, BM.



TREATMENT

I. Prophylactic

1. Early diagnosis: (for high risk groups, e.g. Med & Paramed)
    - Mass radiography.
    - Tuberculin survey.
    - Sputum examination.

2. Immunoprophylaxis: (for high risk groups, e.g. Med & Paramed)
    - BCG vaccination.

3. Chemoprophylaxis: (for contacts of tuberculous patients)
    - INH.

II. Therapeutic

1. General measures

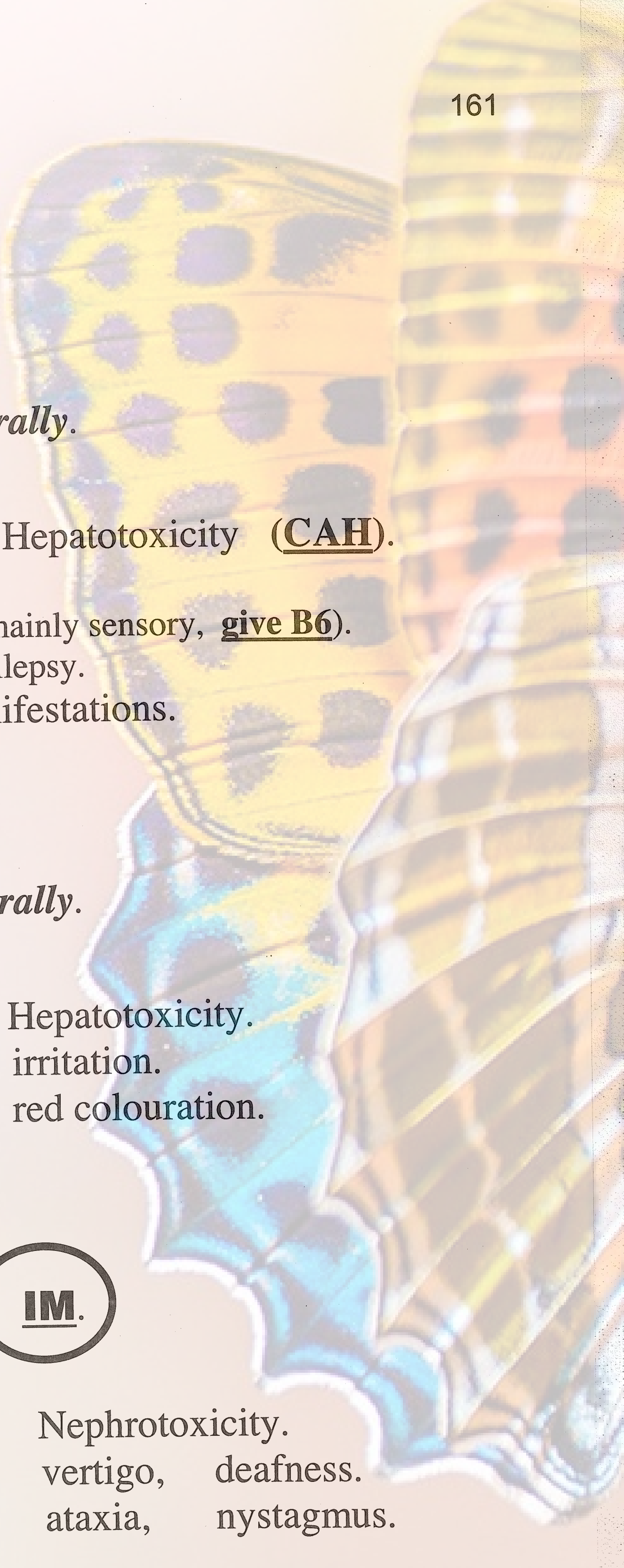
- Proper nutrition.
- Rest in bed: *in acute cases*.
- Drugs: *antipyretics*, *expectorants*, *mucolytics*.

2. Specific measures "Chemotherapy"

A) *Plan of therapy*:

1. Combined drug therapy is needed to:
    - Overcome natural resistance to anti – TB drugs.
    - Accelerate mycobacterial clearance.

2. Long duration of therapy is needed especially in:
    - Extrapulmonary TB.



B) *__Anti-Tuberculous drugs__:*

1. __Isoniazid__ (INH):

Dose:

- 5 mg / Kg / day ***orally***.

Side effects:

- LIVER: Hepatotoxicity (**__CAH__**).
- NEUROLOGICAL:
  - PN (mainly sensory, **__give B6__**).
  - Psychosis & epilepsy.
- LUPUS – LIKE manifestations.

2. __Rifampicin__:

Dose:

- 10 mg / Kg / day ***orally***.

Side effects:

- LIVER: Hepatotoxicity.
- GIT: irritation.
- URINE: red colouration.

3. __Streptomycin__:

Dose:

- 15 mg / Kg / day **__IM__**.

Side effects:

- RENAL: Nephrotoxicity.
- OTOTOXICITY: vertigo, deafness.
- NEUROLOGICAL: ataxia, nystagmus.

4. __Ethambutol__:

Dose:

- 25 mg / Kg / day ***orally***.

Side effects:

- EYE: optic neuritis.

5. Pyrazinamide:

Dose:

- 30 mg / Kg / day ***orally***.

Side effects:

- LIVER: Hepatotoxicity.
- METABOLIC: Hyperuricemia.

6. Ethionamide:

Dose:

- 1 gm / day ***orally***.

Side effects:

- LIVER: Hepatotoxicity.
- GIT: irritation.

7. Cycloserine:

Dose:

- 1 gm / day ***orally***.

Side effects:

- NEUROLOGICAL: dizziness, depression.

8. Para-aminosalicylic acid (PAS):

Dose:

- 10 gm / day ***orally***.

Side effects:

- GIT: irritation.
- HYPERSENSITIVITY: e.g. *skin rash*.

9. Capreomycin, Kanamycin, Amikacin:

Dose & side effects: as streptomycin.

C) *Regimens*:

1. **Short regimen:** **(9 – 12 months)**
   - INH & Rifampicin: for 9 – 12 months.

     plus:
   - Ethambutol or Streptomycin: for the first 2 months only.
2. **Very short regimen:** **(6 months)**
   - INH & Rifampicin: for 6 months.

     plus:
   - Ethambutol or Streptomycin: for the first 2 months only.

     plus:
   - Pyrazinamide: for the first 2 months only.
3. **Classic regimen:** **(Rarely used now: 15, 18 or 24 months)**
   - INH & Rifampicin: for the whole duration.

     plus:
   - Ethambutol or Streptomycin: for the first 2 months only.

D) *Indications of corticosteroids in TB*:

- Corticosteroids are given **under cover of anti- TB drugs** in:
  1. TB meningitis.
  2. TB serositis (*pericardial, pleural, peritoneal*).
  3. TB adrenal glands (*replacement therapy*).
  4. Fulminant cases, *e.g. miliary TB*.

3. Surgery: (resection)

- Tuberculoma.
- Destroyed tuberculous lobe.

4. Collapse therapy.

List of famous TB victims

Writers and poets

- **Honoré de Balzac**
- **Emily Brontë**
- **Anton Chekhov**
- **Franz Kafka** (1883-1924), German-language novelist best known for his novel *The Metamorphosis*.
- **Molière**
- **Jean-Jacques Rousseau**
- **Voltaire**

Composers

- **Frédéric Chopin** (1810-1849). Historical records indicate episodes of hemoptysis during performances.
- **Niccolò Paganini**

Leaders and politicians

- **Simón Bolívar,** considered the liberator of several South American countries, died in 1830.
- **Louis XIII of France**
- **Louis XVII of France**
- **Napoleon II of France**
- **Eleanor Roosevelt**

Others

- **Alexander Graham Bell**
- **Vivien Leigh**
- **Tom Jones**

BRONCHOGENIC CARCINOMA

INCIDENCE

- The most common malignant tumour in ♂ in most of the world.
  - More common in males *(due to smoking).*
  - More common between 50 & 70 years of age.

PREDISPOSING FACTORS

1) **Cigarette smoking:**
   - 90 % of cases are caused by smoking cigarettes.
   - Quantity X duration = Pack X years *(both are risk factors).*
2) Occupational exposure: (miners)
   - Asbestos, nickel, uranium.
3) Chronic lung inflammation, e.g.
   - Interstitial fibrosis.

PATHOLOGY

Types "Histological classification"

1) **Small cell carcinoma** (oat cell carcinoma) **20** %
   - Highly malignant & rapidly growing.
   - The most common to produce paramalignant syndrome.
2) **Non – Small cell carcinoma**
   - a) Squamous cell carcinoma: **40** %
   - b) Large cell carcinoma: **15** %
   - c) Adenocarcinoma: **25** %
     - *Most common in: asbestos exposure, non-smokers, ♀.*
   - d) Alveolar cell (bronchiolar) carcinoma: **1** %
     - *Low grade carcinoma that presents as an alveolar infiltrate.*

Site

- Bronchogenic carcinoma is divided according to its site of origin into:

	Central (hilar)	Peripheral
Incidence	More common	Less common
Type	Squamous cell carcinoma	Adenocarcinoma Large cell carcinoma
Spread	To mediastinum early	To pleura early
Clinical picture	cough, hemoptysis dyspnea, chest pain	pleurisy
Bronchoscope	Easily visible	Hardly visible

- Pancoast tumour is an apical peripheral carcinoma.

SPREAD

1) **Direct**:

- Other parts of the lung.
- Pleura & mediastinum.
- Ribs & vertebrae.

2) **Lymphatic**:

- Hilar & mediastinal lymph nodes.
- Cervical, axillary, abdominal lymph nodes.

NB Retrograde spread along the lung lymphatics may occur leading to lymphangitis carcinomatosis which may lead to subacute corpulmonale.

3) **Blood**:

- Liver.
- Brain.
- Bones.

CLINICAL PRESENTATIONS

1) ASYMPTOMATIC PRESENTATION

- There are no symptoms.
- A coin shadow may be discovered accidentally on: **CXR**.

2) CHEST PRESENTATIONS

A) *Broncho-pulmonary presentations*

1. Cough & hemoptysis.
2. Bronchial obstruction:
    - a- Partial:
        - Localised wheeze persistent after cough.
        - Localised bronchiectasis.
    - b- Complete:
        - Collapse: which may be complicated by lung abscess.
3. Pneumonia: *unresolving, or recurrent.*
4. Lung abscess: *due to:*
    - Secondary infection in a collapsed area.
    - Breakdown of the tumour.

B) *Thoracic inlet or superior sulcus syndrome*

- In cases of pancoast tumour, & manifestations are due to involvement of:
    1. The lower trunk of the brachial plexus, causing:
        - Pain along the medial aspect of the arm, forearm & hand.
        - Weakness & wasting of the small muscles of the hand.
    2. The sympathetic chain, causing:
        - Horner's syndrome.
    3. The blood vessels at the root of the neck, causing:
        - Arterial obstruction: Unequal pulse & BP in the ULs.
        - Venous obstruction: Congested non-pulsating NVs, and dilated veins on the chest wall.

C) *Pleural presentations*

1. **Dry pleurisy**:
   - Due to irritation or infiltration of the pleura.

2. **Pleural effusion**: "of different types"
   a. Exudate (Malignant effusion) ***the most common type***
      - *Massive, bloody.*
      - *Rapidly re-accumulates after aspiration, contains malignant cells.*
      - *May be: mediastinal shift to the same side (due to underlying collapse).*

   b. Transudate:
      - Due to obstruction of the SVC or the azygos vein.

   c. Chylous effusion:
      - Due to obstruction of the thoracic duct.

   d. Suppurative effusion: (Empyema)
      - Due to rupture of lung abscess into the pleura.

D) *Mediastinal presentations*

- The patient may present with features of "Mediastinal syndrome" due to:
  - Direct effect of the tumour.
  - Mediastinal lymph node metastasis.
- **e.g.** Phrenic nerve → Diaphragmatic paralysis, RLN → Hoarseness of voice.

3) METASTATIC PRESENTATIONS

a. Direct metastasis:
- o Mentioned before.

b. Lymphatic metastasis:
- o Mentioned before.

c. Blood metastasis:
- o Liver: enlarged, tender, hard, irregular.
- o Brain: ↑ ICT & focal neurological manifestations.
- o Bones: bony pains & pathological fractures.

4) NON-METASTATIC PRESENTATIONS

“ Paramalignant syndrome ”

- **Definition:**
  A group of manifestations that occur as a consequence of the presence of tumour in the body, but not due to the local presence of tumour cells.
- **Incidence:** occur most commonly with **small cell carcinoma**.
- **Underlying mechanisms:** not so clear, however, proposed explanations are:
  - Production of abnormal metabolites by tumour cells (cytokines or hormones).
  - Immune response against the tumour.
- **Manifestations:**

1. ***Neurological:***
   - CNS: Encephalomyelitis, Subacute cerebellar degeneration.
   - PNS:
     - PN: Peripheral neuropathy.
     - NMJ: Myasthenia gravis, Lambert-Eaton syndrome.
     - Muscles: Secondary myopathy, Dermatomyositis.
2. ***Endocrinal:*** “due to production of ectopic hormones”
   - Pseudohyperparathyroidism.
   - Hypoglycemia.
   - Cushing’s syndrome.
   - SIADH
   - Gynecomastia.
3. ***Hematological:***
   - Granulocytosis: due to the production of **G-CSF**.
4. ***Dermatological:***
   - Dermatomyositis.
   - Acanthosis nigricans.
   - Herpes zoster.
   - Hyperpigmentation.
   - Pruritus.
5. ***Others:***
   - Clubbing.
   - Cachexia.
   - Fever not related to infection.

INVESTIGATIONS

1) IMAGING

A) CXR: [*]

- It detects only tumours bigger than 2 cm in size.
- It may show:
  - ***Lung***:
    - Mass: Coin shadow, or big mass, or apical mass (pancoast).
    - Complication: Pneumonia, lung abscess, or collapse.
  - ***Pleura***:
    - Pleural effusion, with mediastinal shift to the same side.
  - ***Mediastinum***:
    - Mediastinal mass: it could be the tumour itself or its LN.
  - ***Diaphragm***: **(Paralysis)**
    - Elevated copula on the affected side.
    - Paradoxical movement on the screen.
  - ***Chest wall***:
    - Erosion of the ribs or vertebrae.

B) CT chest:

- It is very accurate & sensitive in detecting the tumour.
- It can detect small tumours (< 2 cm in size) that do not appear on CXR.

2) BRONCHOSCOPY

- Visualization & localization of the tumour, especially the central type.
- Biopsy: could be taken for pathological examination.
- BAL: could be obtained & examined for malignant cells.

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[*] See different CXR figures of bronchogenic carcinoma

3) CYTOLOGY "for detection of *malignant cells*"

1. Sputum examination: may be positive for *malignant cells*.
2. Aspiration of pleural effusion: may be positive for *malignant cells*.
3. BIOPSY:
    - *Transbronchial:* through bronchoscopy.
    - *Lung biopsy:* open, percutaneous, or thoracoscopy.
    - *Lymph node biopsy:* from enlarged LN.

4) LABORATORY INVESTIGATIONS

- CBC: anemia, granulocytosis.
- ESR: elevated (markedly).
- Investigations for diagnosis of endocrinal disturbances: e.g.
    - *Serum Ca:* *for pseudohyperparathyroidism.*
    - *Serum glucose:* *for hypoglycemia.*
    - *ACTH:* *for Cushing's syndrome.*

DIFFERENTIAL DIAGNOSIS

A) Clinically:

- The possibility of bronchogenic carcinoma: should be considered in any: Old, chronic heavy smoker, male: *with a chest complaint*.

- The DD of bronchogenic carcinoma includes:
    1. Other causes of prolonged cough or hemoptysis.
    2. Other causes of localized bronchial obstruction.
    3. Unresolving pneumonia or lung abscess.
    4. Other causes of pleural effusion.
    5. Other causes of mediastinal syndrome.
    6. **TB**.

B) ***Radiological**ly*:

- From other causes of **solitary pulmonary shadow** *(coin shadow)*:

1. Pulmonary tumours:
    - Benign tumour.
    - Malignant tumour.
    - Solitary metastasis.
2. Pulmonary infections, e.g.
    - Pneumonic patch.
    - TB.
3. Pulmonary infarction.
4. Foreign body.

TREATMENT

- **Successful surgery offers the only chance of complete cure.**
- **Choice of treatment depends on:**
    1. Histological type of the tumour.
    2. Operability:
        - o No evidence of hilar or mediastinal involvement.
        - o No evidence of extra-thoracic spread.
- **Lines of treatment include:**
    1. For non-small cell carcinoma:
        - A) Operable:
            - Pneumonectomy or lobectomy: may be followed by postoperative irradiation.
        - B) Non-operable:
            - Chemotherapy & palliative radiotherapy.
    2. For small cell carcinoma:
        - Chemotherapy: **e.g.** cyclophosphamide.
        - Radiotherapy: for treatment of the primary tumour & prophylaxis against metastases.

Neurological manifestations of bronchogenic carcinoma

1) CHEST PRESENTATIONS

Thoracic inlet or superior sulcus syndrome:

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1. The lower trunk of the brachial plexus, causing:
    - Pain along the medial aspect of the arm, forearm & hand.
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2. The sympathetic chain, causing:
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Mediastinal presentations:

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- RLN → Hoarseness of voice.

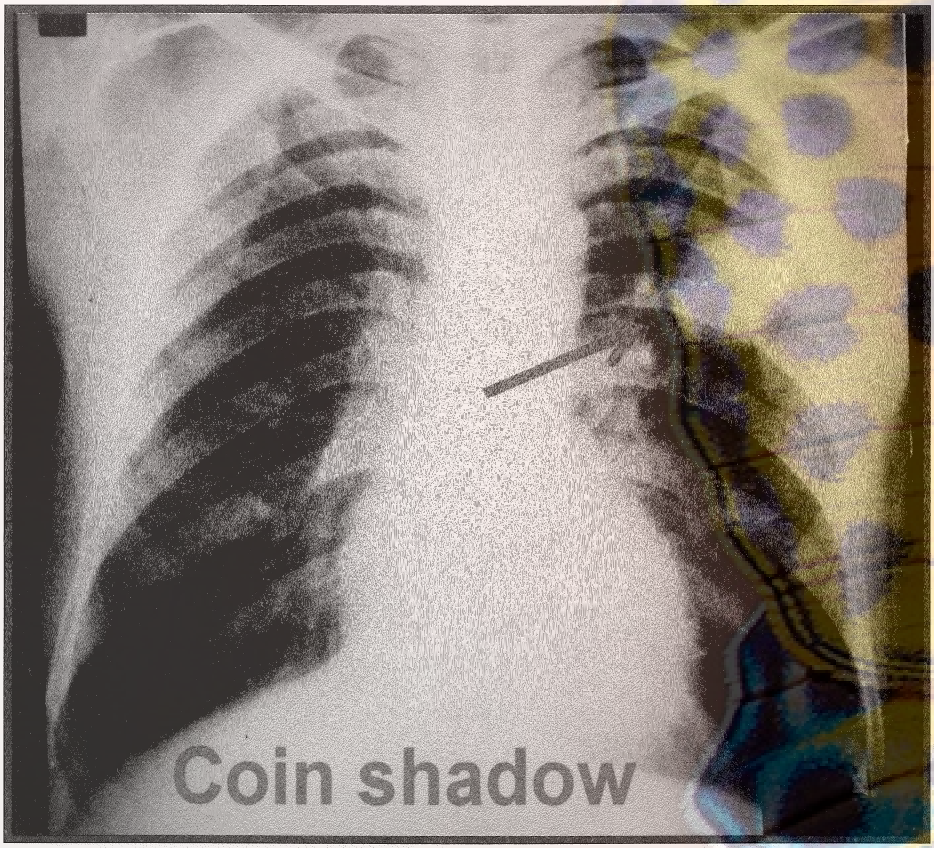
2) METASTATIC PRESENTATIONS

- Brain: ↑ ICT & focal neurological manifestations.

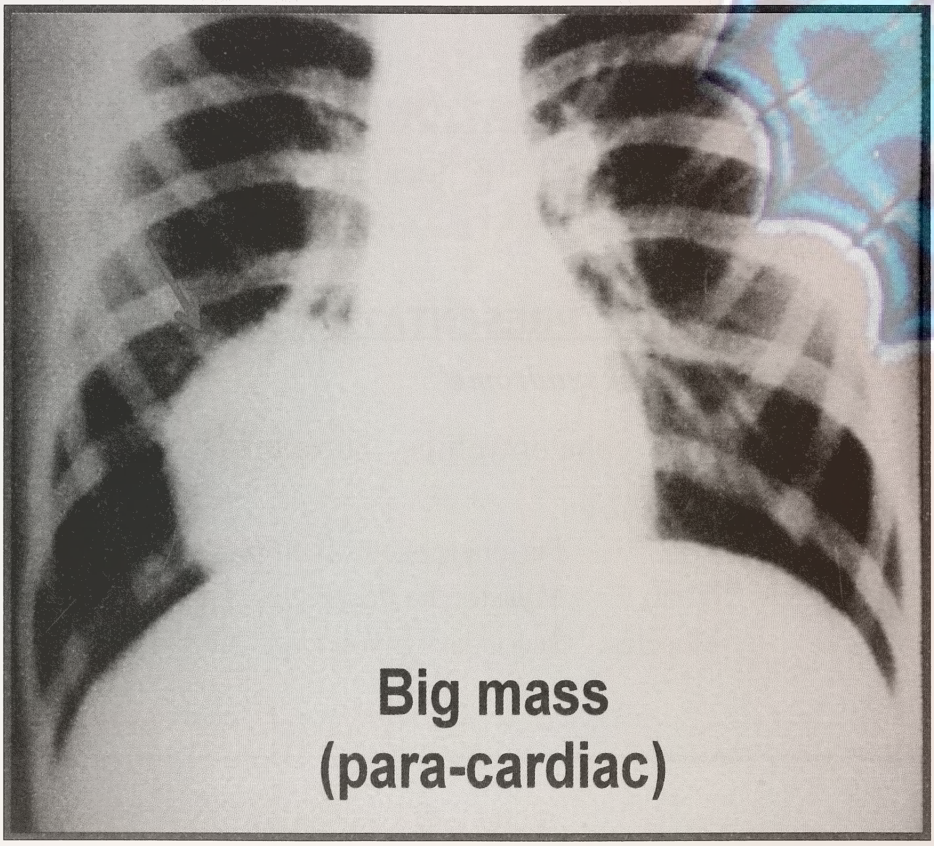
3) NON-METASTATIC PRESENTATIONS

"Paramalignant syndrome"

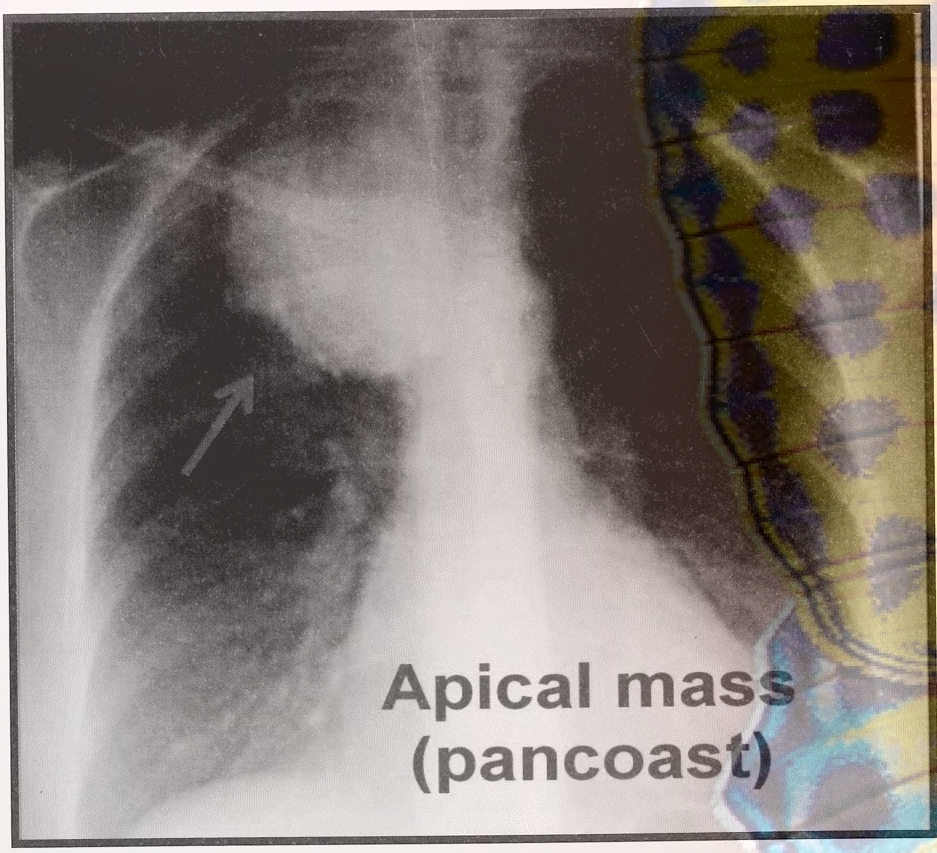
- CNS: Encephalomyelitis, Subacute cerebellar degeneration.
- PNS:
    - PN: Peripheral neuropathy.
    - NMJ: Myasthenia gravis, Lambert-Eaton syndrome.
    - Muscles: Secondary myopathy, Dermatomyositis.



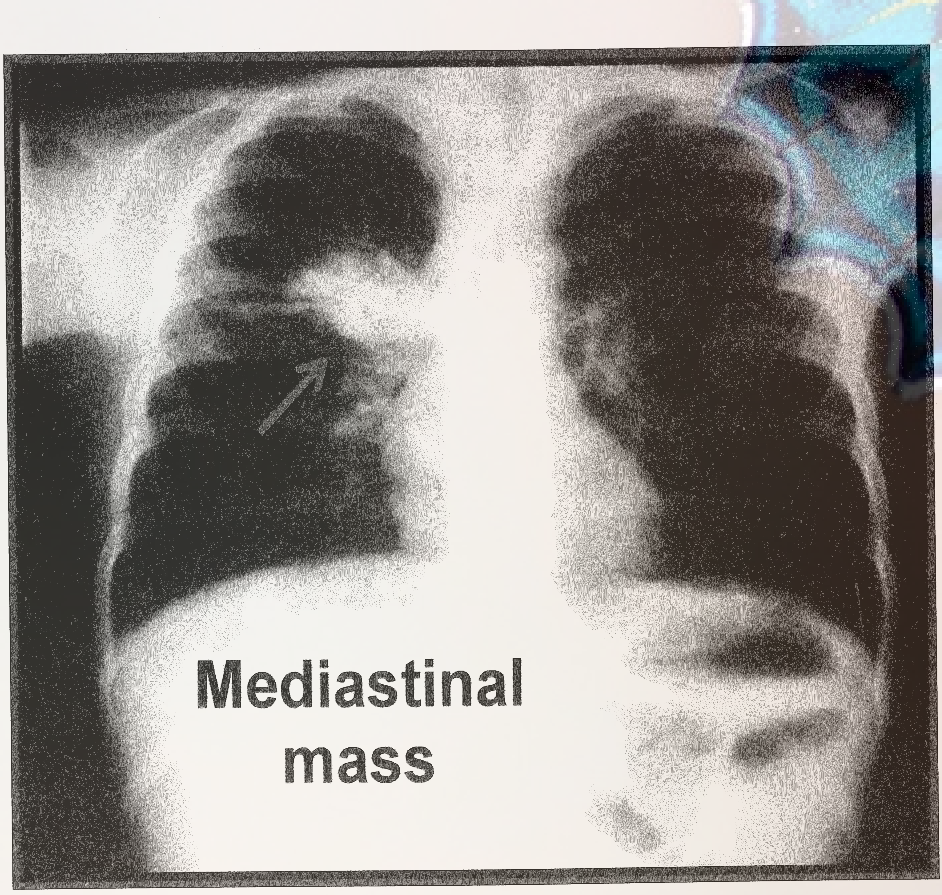
Coin shadow



Big mass
(para-cardiac)



Apical mass (pancoast)



Mediastinal mass

List of famous Bronchogenic carcinoma victims

- Walt Disney
- Yul Brynner

ANATOMY OF THE MEDIASTINUM

- **The mediastinum is the central part of the thorax.**

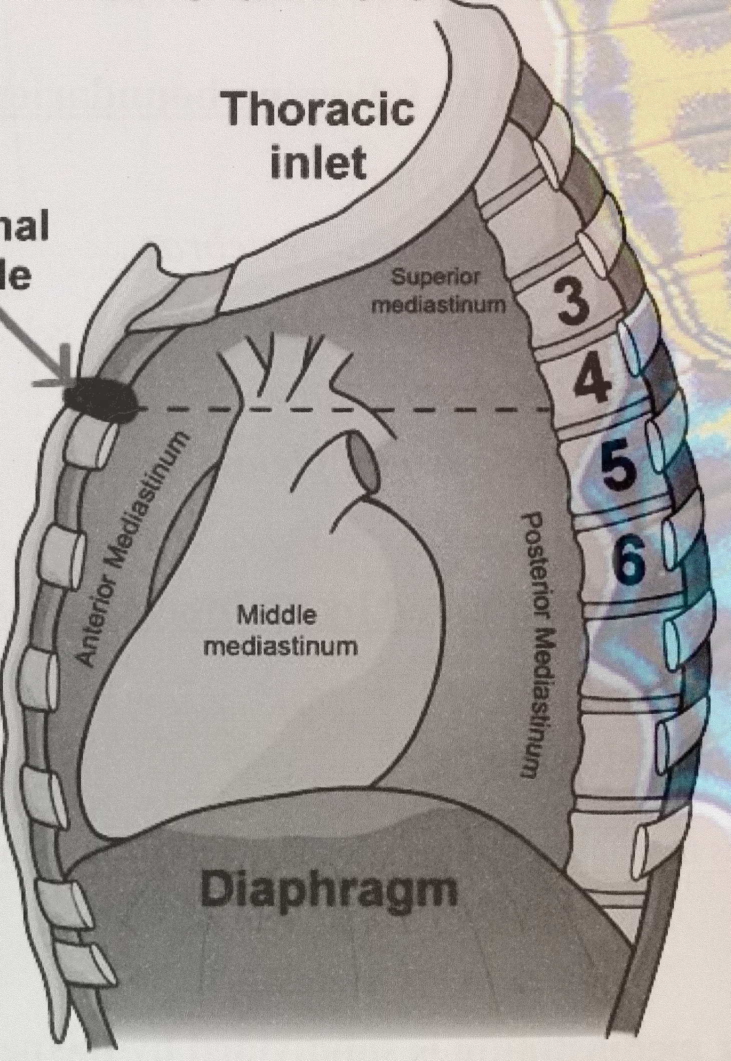
- **The mediastinum has the following boundaries:**
  - *Anteriorly:* *the sternum.*
  - *Posteriorly:* *the thoracic vertebrae.*
  - *Superiorly:* *the thoracic inlet.*
  - *Inferiorly:* *the diaphragm.*
  - *Laterally:* *the parietal pleura.*

- **The mediastinum is divided by an imaginary line into:**
  - Superior mediastinum.
  - Inferior mediastinum.

The line passes from the sternal angle to the lower border of the 4th thoracic vertebra

- **The inferior mediastinum is subdivided by the pericardial sac into:**
  - Anterior mediastinum.
  - Middle mediastinum.
  - Posterior mediastinum.

Boundaries


Thoracic inlet
Sternal angle
Superior mediastinum
3
4
5
6
Anterior Mediastinum
Middle mediastinum
Posterior Mediastinum
Diaphragm

CONTENTS OF THE MEDIASTINUM

I. Superior mediastinum:

1. *Vessels:*

- Arteries: Arch of the aorta & its 3 big branches.
- Veins: SVC & the 2 innominate veins.

2. *Tubes:*

- Upper parts of: *oesophagus*, *trachea*, *thoracic duct*.

3. *Nerves:*

- Vagus & Left RLN.
- Upper sympathetic ganglia.

Autonomic nerves

- Phrenic nerves.

II. Anterior mediastinum:

1. Thymus gland.
2. Fat.

III. Middle mediastinum:

1. Pericardial sac & its contents (*heart & its big vessels*).
2. Trachea & the main bronchi.
3. Phrenic nerves.

IV. Posterior mediastinum:

1. *Vessels:*

- Arteries: Descending aorta.
- Veins: Azygos & hemiazygos veins.

2. *Tubes:*

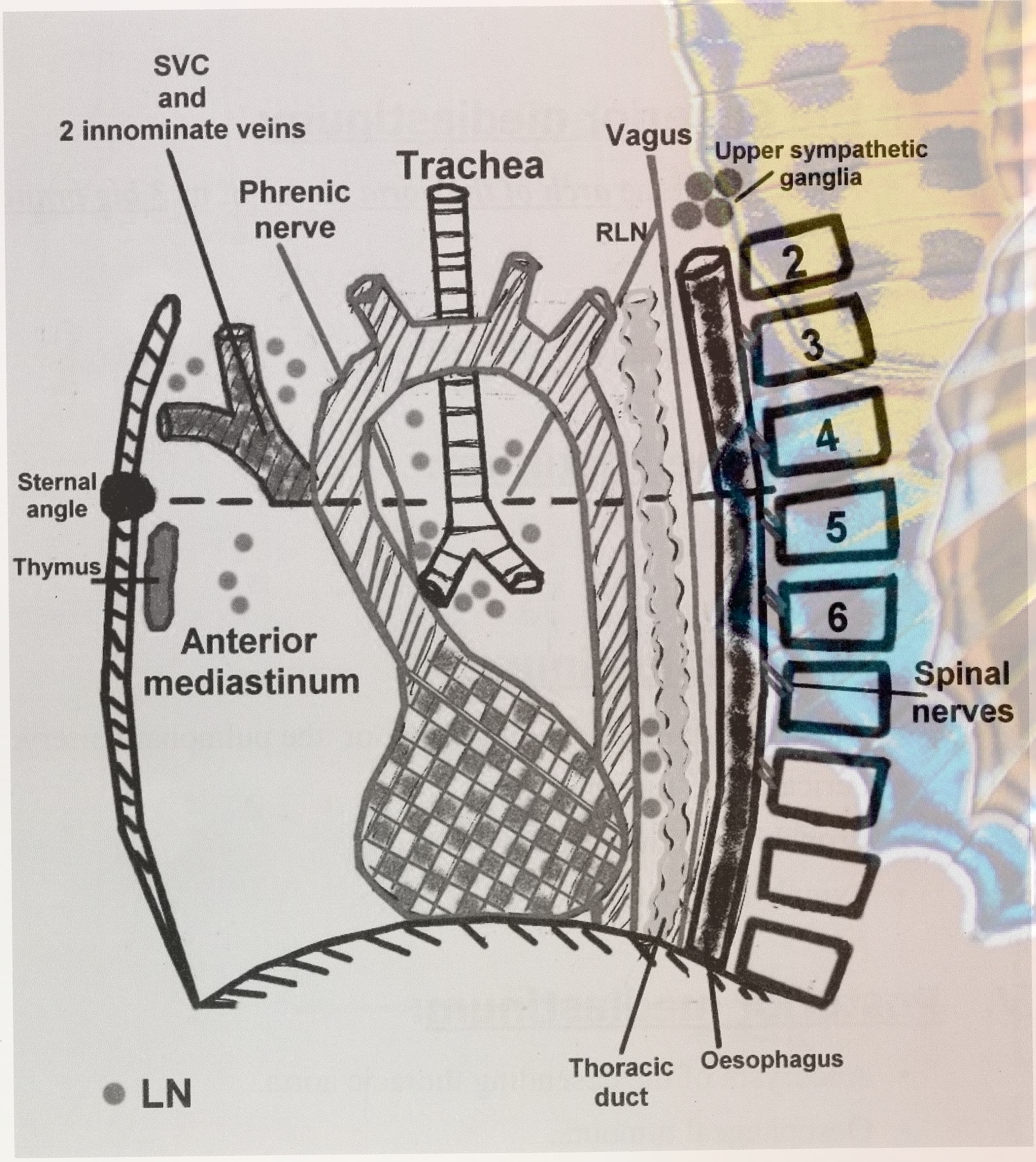
- Oesophagus.
- Thoracic duct.

3. *Nerves:*

- Vagus nerve.
- Spinal nerves.

NB ***Lymph nodes are present in different parts of the mediastinum.***

Illustrative anatomy of the mediastinum


SVC
and
2 innominate veins
Trachea
Phrenic nerve
Vagus
Upper sympathetic ganglia
RLN
2
3
4
5
6
Sternal angle
Thymus
Anterior mediastinum
Spinal nerves
Thoracic duct
Oesophagus
LN

MEDIASTINAL SYNDROME

ETIOLOGY

I. In the Superior mediastinum:

- Aneurysm of the ***arch of the aorta*** or one of its ***3 big branches***.
- Tumours.
- Retrosternal goitre.
- Lymphadenopathy.

II. Anterior mediastinum:

- Thymoma.

III. Middle mediastinum:

- Aneurysm of the ascending aorta or the pulmonary artery.
- Pericardial effusion.
- Bronchial carcinoma.
- Lymphadenopathy.

IV. Posterior mediastinum:

- Aneurysm of the desending thoracic aorta.
- Oesophageal tumours.
- Hiatus hernia.
- Neurofibroma.

CLINICAL PICTURE

I. Manifestations of the cause.

II. Pressure manifestations:

1. *Pressure on vessels:*

A) Branches of aortic arch:

- Unequal pulse & BP in the upper limbs.

B) SVC:

- Neck veins: *congested non – pulsating*.
- Dilated veins: on the chest wall filling from above downwards.
- Oedema & cyanosis: *face*, *neck* & *upper limbs*.

2. *Pressure on tubes:*

A) Oesophagus:

- Dysphagia.

B) Trachea & bronchi:

- Dyspnea, stridor,
- Cough *(brassy)*.
- Wheeze *(localised)*.

C) Thoracic duct:

- Pericardial effusion.
- Pleural effusion.
- Peritoneal effusion (ascites).

} **Chylous**

3. ***Pressure on nerves:***

A) Vagus or left RLN:
- Hoarseness of voice.

B) Sympathetic chain:
- Horner's syndrome.

} **Autonomic nerves**

C) Phrenic nerve:
- Diaphragmatic paralysis.

D) Brachial plexus: (lower trunk)
- Pain along the medial aspect of: *arm, forearm, hand.*
- Weakness & wasting of: *small muscles of the hand.*

E) Intercostal nerves:
- Intercostal neuralgia.

4. ***Pressure on bones:*** **(Ribs, sternum, spine)**

- Bone pains.

INVESTIGATIONS

I. Imaging:

1. CXR:

- To detect the mediastinal mass (size & site).

2. CT & MRI chest:

- More accurate in diagnosing the mass.

3. Fluoroscopy:

- To detect the position & mobility of the diaphragm.
- To detect pulsations of an aneurysm.

4. Others:

- Echocardiography, Ba swallow, thyroid scan, lymphangiography.

II. Endoscopy:

- Bronchoscopy.
- Oesophagoscopy.
- Mediastinoscopy.

III. Exploratory thoracotomy.

TREATMENT

- **TTT of the cause.**